A STUDY OF ECHOCARDIOGRAPHIC EPICARDIAL ADIPOSE TISSUE THICKNESS AND CAROTID INTIMA MEDIA THICKNESS TO PREDICT SEVERITY OF CORONARY ARTERY DISEASE

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CERTIFICATE

This is to certify that the dissertation entitled – **"A STUDY OF ECHOCARDIOGRAPHIC EPICARDIAL ADIPOSE TISSUE THICKNESS AND CAROTID INTIMA MEDIA THICKNESS TO PREDICT SEVERITY OF CORONARY ARTERY DISEASE"** is the bonafide original work of **Dr. P. VINODH KUMAR** in partial fulfillment of the requirements for D.M. Branch II (CARDIOLOGY) examination of THE TAMILNADU DR.M.G.R. MEDICAL UNIVERSITY to be held on August 2014. The period of post graduate study and training was from August 2011 to July 2014

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DECLARATION

I **Dr. P. VINODH KUMAR**, solemnly declare that this dissertation entitled – **"A STUDY OF ECHOCARDIOGRAPHIC EPICARDIAL ADIPOSE TISSUE THICKNESS AND CAROTID INTIMA MEDIA THICKNESS TO PREDICT SEVERITY OF CORONARY ARTERY DISEASE"** is the bonafide original work done by me at the Department of Cardiology, Stanley Medical College and Government Stanley Hospital during the period 2011-2014 under the guidance and supervision of the Professor and Head of Department of Cardiology of Stanley Medical College and Government Stanley Hospital, **Prof. Dr. K.KANNAN, M.D., D.M., FACC.** This dissertation is submitted to THE TAMILNADU DR.M.G.R. MEDICAL UNIVERSITY, towards partial fulfillment of requirement for the award of D.M. Degree (Branch - II) in cardiology.

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ABBREVIATIONS

AHA	-	American Heart association
AWMI	-	Anterior Wall Myocardial Infarction
AV	-	Atrioventricular
BMI	-	Body Mass Index
CABG	-	Coronary Artery By-Pass Graft
CAC	-	Coronary Artery Calcium
CAD	-	Coronary Artery Disease
CAG	-	Coronary Angiogram
CHF	-	Congestive Heart Failure
CIMT	-	Carotid Intima Media Thickness
CMR	-	Cardiac Magnetic Resonance
CSA	-	Chronic stable angina
CT	-	Computed tomography
DVD	-	Double Vessel Disease
EA	-	Effort Angina
EAT	-	Epicardial Adipose Tissue
EATT	-	Epicardial Adipose Tissue Thickness
FFA	-	Free Fatty Acid
FMD	-	Flow Mediated Dilatation
HAART	-	Highly Active Antiretroviral Therapy
HDL	-	High Density Lipoprotein
ICCU	-	Intensive Coronary Care Unit
IL	-	Interleukin
IR	-	Insulin Receptor
IWMI	-	Inferior Wall Myocardial Infarction
LAD	-	Left Anterior Descending artery
LDL	-	Low Density Lipoprotein
LM	-	Left Main coronary artery

LPL	-	Lipoprotein lipase
LV	-	Left Ventricle
LVEF	-	Left Ventricular Ejection Fraction
LCX	-	Left Circumflex artery
MDCT	-	Multi Detector Computed Tomography
MeTs	-	Metabolic Syndrome
MI	-	Myocardial Infarction
MR	-	Mitral Regurgitation
MRI	-	Magnetic Resonance Imaging
NAFLD	-	Non Alcoholic Fatty Liver Disease
RCA	-	Right Coronary Artery
RV	-	Right Ventricle
RVOT	-	Right Ventricle Outflow Tract
SD	-	Standard Deviation
SMC	-	Smooth Muscle Cell
SVD	-	Single Vessel Disease
TNF	-	Tumour Necrosis Factor
TVD	-	Triple Vessel Disease
WC	-	Waist circumference
WHR	-	Waist Hip Ratio

INTRODUCTION

Coronary artery disease (CAD) is considered the leading cause of death in the world. In 2004, CAD was the cause of 7.2 million deaths (About 12% out of a total of about 60 million deaths) [1]. At the same time in India, CAD related deaths was the most common cause, leading to about 1.5 million deaths (14% out of a total of approx.10 million deaths).

CAD now causes more death and disability in socioeconomically low- and middle-income countries, such as India. The CAD related death rate is increasing alarmingly when compared to high-income countries. CAD in low- and middle-income countries affects people at younger ages, compared to high-income countries [2]. Unadjusted CAD rates in India have ranged from about 1% to about 13%, in urban populations and about 1.6% to 7.4%, in rural populations [3]. In a study from urban population like Chennai the prevalence of CAD was 11% [4].

This alarming increase in CAD prevalence is a cause of concern. While the major aspect in dealing with CAD is identification and modification of risk factors, equally important is the ability to identify the individuals in early stage of CAD, before development of any adverse clinical event, chronic disability or death.

Subclinical atherosclerosis has been shown to develop years before manifestations of cardiovascular diseases are seen clinically. Hence proper identification of predictors or markers of premature atherosclerosis is very crucial. Patients with increased visceral fat have also been found to be at increased risk for cardiovascular events.

Epicardial fat or Epicardial adipose tissue (EAT) represents a true visceral fat and has been proposed as a cardiometabolic risk factor [5]. Epicardial fat is the adipose tissue present between the myocardium and the visceral pericardium and it has been credibly shown to be a true visceral fat and with all the specific traits of insulin resistant states [6]. Epicardial adipose tissue thickness (EATT) has clinically correlated with abdominal visceral fat, metabolic syndrome, subclinical atherosclerosis, coronary artery disease and cardiac morphology.

In a study evaluating EATT in hypertensive patients found that a cut off of 3.1 mm can be used in predicting metabolic syndrome with 100% sensitivity and 79% specificity [7]. Another small study evaluating EATT in patients with acute coronary syndrome undergoing coronary angiogram, it has been shown that the mean EATT thickness was 5.5 ± 0.5 mm [8]. A larger study by S.G. Ahn et al showed that a value of 3.0 mm was an independent factor for predicting CAD [9]. However a recent study which attempted to establish echocardiographic cut off points for diagnosis of

CAD had shown a much higher value of more than 10mm for predicting a coronary stenosis [10]. These previous studies showed wide variations in the mean EAT and the ability to predicting CAD. Hence there is a need to study the correlation between EAT and the extent of coronary artery involvement and also establish a reliable range of values for predicting CAD.

Carotid intima media thickness (CIMT) has been recently proposed as a dependable surrogate marker for coronary atherosclerosis. An increased cross-sectional carotid intima-media thickness was associated with significantly high levels of established cardiovascular risk factors and atherosclerosis elsewhere in the arterial system. Carotid IMT has been shown by many observational studies to be a excellent indicator of the presence and severity of coronary artery disease [11, 12]. Studies have shown that CIMT is very useful in screening patients with increased risk for adverse events including stroke, CAD and MI [13, 14, 15].

EATT and CIMT have been compared in various disease populations including diabetes mellitus, metabolic syndrome, hypertensive patients to assess the vascular function and HIV patients taking HAART. However there are no studies to compare or correlate EATT and CIMT to the extent of coronary artery disease by coronary angiography and in the ability to predict the coronary artery disease.

AIMS AND OBJECTIVES OF THE STUDY

1. To assess the relation between EATT and the severity of coronary artery disease.
2. To correlate Epicardial adipose tissue thickness and carotid intima media thickness in predicting severity of coronary artery disease.
3. To assess the incremental benefit of combination of EATT and CIMT to predict the severity of coronary artery disease.
4. To analyse the feasibility of EATT as a non invasive screening test to detect CAD.

REVIEW OF LITERATURE

Atherosclerosis is a complex disease with a multifactorial etiology related to inheritance and traditional and non-traditional risk factors. Atherosclerosis progresses through various stages, proceeding from subclinical to clinically significant stages. Studies has shown that hyperlipidemia induced macrophage infiltration is the earliest pathologic change [16].

The atherosclerotic lesions have been extensively studied at autopsy and specific morphologies have been assigned to categories established by American Heart Association consensus group as follows [17].

Non progressive Atherosclerosis:

1. The earliest vascular change seen microscopically is adaptive intimal thickening – AHA type I.
2. the next type is fatty streaks which represent non raised lesions consisting of intra and extra cellular lipid deposits – AHA type II.

Progressive Atherosclerosis:

1. Pathological intimal thickening which consist of lipid pools located near medial wall, also known as intermediate lesion – AHA type III [18].

2. The first of advanced lesion are considered fibroatheromas, characterized by acellular necrotic core – AHA type IV.
3. The fibrous cap is distinct from the necrotic core – AHA type V.
4. Complicated plaques with thin cap with surface defect and/or hematoma-haemorrhage, and/or thrombosis – AHA type VI [18].

VISCERAL FAT AND ITS ROLE IN ATHEROSCLEROSIS

Increased upper body fat observed commonly in males and measured by an higher waist-to-hip ratio (WHR) and it has been demonstrated to be an good predictor for morbidity and mortality from coronary heart disease, cancers, and diabetes mellitus [19, 20]. This visceral fat is also strongly associated with hyperinsulinemia, glucose intolerance, and hypertriglyceridemia [21]. Upper-body fat consists of visceral (inner) and subcutaneous (outer) depots. The visceral fat is seen within the body cavity and it is enveloping the internal organs. It is consists of the mesenteric fat and also the greater and lesser omental fat depots, with each of them having distinct function [22]. Visceral fat represents 6% of total body fat in women and 20% of total body fat in men.

MECHANISM OF METABOLIC DERANGEMENT:

Regional differences in metabolic properties of adipocytes: Dietary fat labeled with radioactive isotopes when given to those undergoing elective

surgery, have shown a considerable high formation of triglyceride in upper segment of body compared to lower segment body fat depots [23]. Visceral fat cells compared to subcutaneous derived adipocytes have demonstrated higher level of catecholamine-induced lipolysis [24]. Visceral fat depots have higher number of beta 1- and 2-adrenergic receptors and have shown better lipolytic program compared to subcutaneous cells [25]. The Anti lipolytic effect of insulin is significantly less in visceral adipocytes [26]. This may be related to reduced insulin receptor (IR) affinity and also due to visceral fat having significantly lesser levels of insulin receptor substrate -1 (IRS-1) protein [26].

Effect of Steroid Harmones:

Visceral adipocytes express a greater number of glucocorticoid receptors and activated glucocorticoid receptors present in adipocytes causes an increase in lipoprotein lipase (LPL) activity and thereby enhancing triglyceride storage [27]. However the roles of sex steroids are complex and not clearly understood. Esterogen enhances LPL activity and androgen reduces it.

Production of hormones, polypeptides and cytokines:

Central obesity is connected to atherothrombotic events. PAI-1 has been shown to be produced and secreted from adipocytes. Compared to subcutaneous cells, visceral adipocytes have demonstrated to produce

significantly more of this peptide than did subcutaneous cells and also it has been found that the levels of PAI-1 correlated robustly with visceral fat mass [28]. Studies have clearly demonstrated that obese patients have high levels of adipose TNF mRNA and proteins and weight loss produces a decrease in TNF levels [29]. The adipocytes and also by stromal cells in the fat depot also secrete IL-6. Like TNF, IL-6 inhibits formation of LPL, but unlike TNF it does not enhance lipolysis [30].

IMAGING OF VISCERAL FAT:

CT has been considered the gold standard for imaging visceral fat since its fast acquisition rate prevents any effect on the region of interest caused by the effect of bowel movement. However there are the hazards of ionising radiation. MRI has evolved into a dependable and a safer alternative. Studies have shown that utilising MRI 0.3 to 1.5T is reliable and accurate in estimating visceral fat [31, 32]. Tanaka et al [33] has reported that 1.5T MRI caused underestimation of the area and volume of visceral fat. A recent study with 3.0T MRI has demonstrated that it was able to delineate and quantify visceral fat as good as CT and better than MRI with lesser tesla [34].

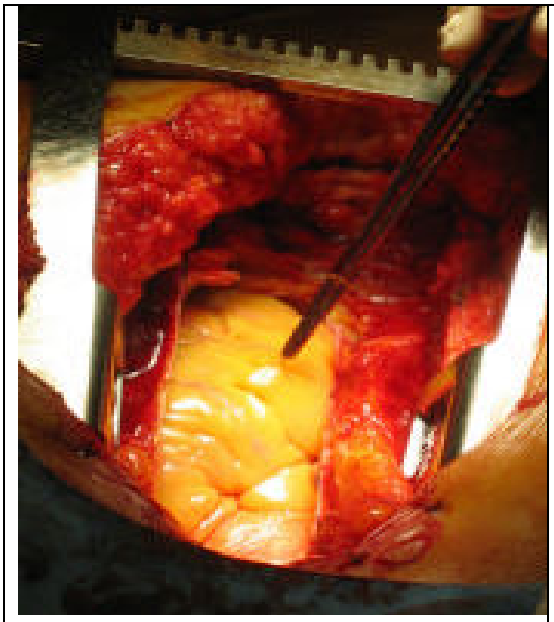
EPICARDIAL ADIPOSE TISSUE

Relationship between Epicardial adipose tissue and visceral adiposity:

The relationship between intraabdominal fat deposit and pericardial fat deposit especially the epicardial fat has been clearly demonstrated. Iacobellis et al., demonstrated by transthoracic echo and by gated MRI that EAT has excellent correlation with MRI abdominal VAT [5]. Studies using CT have also shown that the EAT volume correlated significantly with the abdominal visceral fat [35, 36].

STRUCTURE AND FUNCTION OF EPICARDIAL ADIPOSE TISSUE

Epicardial tissue is situated over the surface of the heart i.e. in direct contact over the myocardium. It is confined to the visceral pericardium and is found commonly around the proximal part of coronary vessels and within the Atrioventricular and interventricular grooves [37]. The fat which surrounding the heart but present outside the pericardium is called paracardiac fat.



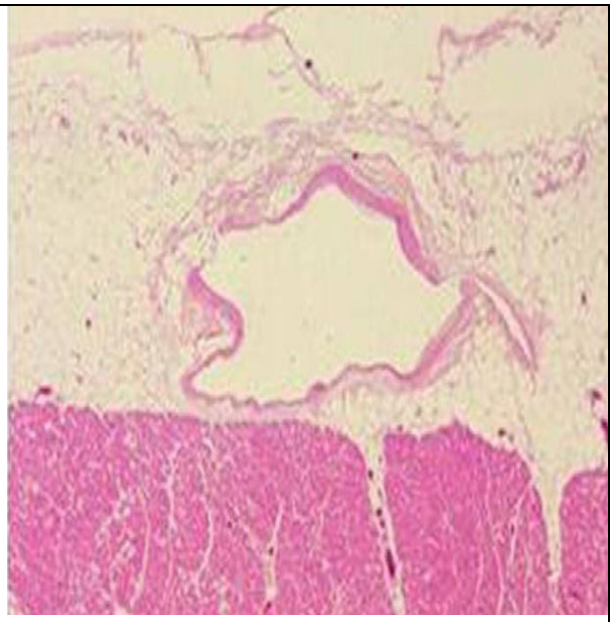


FIGURE1: Macroscopic and microscopic imaging of epicardial fat

The EAT and paracardiac fat have different embryological origin [38]. The splanchnopleuric mesoderm gives rise to the EAT and the mesenteric and the omental fat deposits, where as the primitive thoracic mesenchyme gives rise to paracardiac fat and the parietal pericardium. The vascular supply for the EAT is from the coronary arteries and the blood supply for the paracardiac fat is from the pericardiophrenic branches from the internal mammary arteries.

Plausible physiological role of EAT:

The EAT and other visceral fat deposits have a significant higher capacity for uptake and release of FFA and also lesser utilization of glucose [39]. This function can act as a buffer by preventing high level of FFA by scavenging

excess FFAs in the myocardial microcirculation. The EAT may act as source of energy during periods of ischemia because of its high lipolytic activity. EAT also contains the ganglia and the interconnecting neural plexuses which are part of the cardiac nervous system. These neural plexuses play a major role in mediating angina pain during coronary occlusion. EAT can have a protective effect on these ganglia and plexuses [38]. The other possible role is its cushion effect buffering the coronary artery from torsion caused by the arterial pulse wave and cardiac contraction [40].

EAT role as endocrine and paracrine organ:

EAT has been shown to be an effective endocrine organ which may have major implications on cardiac functions. EAT secretes inflammatory mediators like IL-1, IL-6, TNF-α and MCP-1 in patients with increased with increased cardiac risk [41]. The cytokines secreted in the epicardial fat can diffuse though the coronary wall from outside. These cytokines are thought to have effect on the vascular tone and cellularity [41]. Adiponectin and adrenomedullin secreted by EAT may have cardioprotective effects through anti-inflammatory and anti atherogenic properties [42]. In Chronic CAD has shown to down regulate protective adipokines and through hemodynamic regulation by CABG has shown to increase adrenomedullin [43]. In patients with CAD the epicardial fat showed c-Jun N-terminal kinase (JNK) activity and NFkappaB. The presence of

activated macrophages in CAD is suggested by an increased level of TLR-2 and TLR-4 and also by increased systemic lipopolysacharides [44].

EAT and association with obesity:

It has been shown by studies using MDCT that volume of EAT correlated consistently with the extent of obesity. However, most studies have demonstrated that it correlated significantly only with intra-abdominal fat and showed only moderate correlation with BMI which considered an index of general obesity and also with waist circumference which an index of central obesity [6, 36]. EATT by echo has shown a significant correlation with waist circumference [6].

EAT and relation to metabolic syndrome:

Studies have suggested that abdominal visceral fat is an independent predictor of metabolic risk [45, 46].With MDCT the peri-coronary EATT had strongest correlation with glucose parameters, modest with lipid parameters, and least with blood pressure [28, 36]. EAT volume measured by CT strongly associated with lipid parameters [47]. EAT by echo has significant correlation with diastolic blood pressure and fasting insulin. It also correlated with LDL cholesterol, blood glucose, high-density lipoprotein cholesterol, and systolic blood pressure [6]. Metabolic syndrome has noted to occur in patients with normal waist circumference (< 102 in males and < 88 in females) and normal BMI (< 25). In a study on 172

patients of more than 40 years of age with normal BMI and waist circumference it was found that EAT was significantly elevated with MeTs than those without MeTs. Also it was found that prediction of MeTs was effectively improved when combining EATT with BMI and waist circumference [7].

EAT and its association with myocardial structure and function:

Studies with proton magnetic resonance spectroscopy have shown that the endogenous triglyceride depot of the heart is less than 1.0% of total cardiac mass in healthy individuals [48]. Cardiac steatosis is characteristic finding in patients with type 2diabetes and obesity. Compared to controls myocardial triglyceride levels are increased by about two to four fold in these groups. This myocardial adiposity is associated with increased LV mass, reduced septal thickening and impaired LV diastolic function [49]. Studies using echocardiography have demonstrated that EATT was strongly associated with increased left ventricular mass, LA size, and diastolic dysfunction [50]. A sub study from the Framingham study had shown that except for the left atrial dimension the other parameters did not have an independent correlation with EAT thickness [51].

EAT and its association with Coronary atherosclerotic disease:

Epicardial adipose tissue and its role in the coronary atherosclerosis disease have been extensively evaluated. As early as 2001,

Taguchi and colleagues demonstrated that the pericardial fat (combined epicardial and paracardiac fat) determined by CT when compared to visceral abdominal fat was a strong independent risk variable for CAD [52]. A study on the follow up participants without any cardiovascular disease from the Framingham study demonstrated that though pericardial fat correlated with cardiovascular risk factors and other measures of adiposity, it turned out that VAT had a more significant correlation for most of the metabolic risk factors. However, pericardial fat has been shown to be independently associated with coronary calcification, which has been suggested to be due to the local toxic effects of that these fat depots [35].

The first study on EAT thickness and its correlation with CAD severity was done by Jeong et al [53]. In this study echocardiographic evaluation of epicardial fat with correlation with angiographic CAD severity showed that the patients with EAT thickness more than 7.6mm had a significantly higher gensini score. Another larger study by Ahn SG et al., involving 527 patients also demonstrated that EAT as defined by echocardiography is higher in patients with CAD than in those without CAD (4.5mm Vs 1.5mm). This study also showed that the thickness of EAT was higher in patients with unstable angina when compared against patients with chronic stable angina or patients having atypical chest pain (4.5mm, 3.0mm and 1.5mm respectively) [9]. Other studies of EAT

estimation based on echocardiography have also demonstrated the relation between the increased EAT thickness and the presence and extent of CAD [54, 55]. However the Echocardiographic cut off point for reliably establishing the presence and also in predicting the severity of CAD varied significantly. Feyter *et al*., found that the mean EAT was 10.91±1.9 mm in the patients who are undergoing coronary angiography [55]. Mehrnoush Toufan et al have tried to establish a cut off point for EAT [10]. They found that RV EAT≥ 10 mm and RV apex EAT ≥ 8 mm had sensitivity and PPV of more than 70% in predicting coronary stenosis ≥ 50% and RVOT EAT ≥ 13 mm is of PPV=83.5% for predicting coronary stenosis ≥ 50%.

The role of EAT has also been evaluated in patients with acute coronary syndrome. The study by SG Ahn et al found that compared to patients with patients with stable angina pectoris (3.0mm) the patients with unstable angina had a higher EAT thickness (4.5mm). The study of Toufan M et al which had 51.2% of the study patients undergoing coronary angiogram had ACS, found that 79.2% patients had a RVOT EAT ≥ 13mm and 77% patients had a RV EAT ≥ 10mm. In a small study of 65 ACS patients, it was found that EAT had a positive correlation with troponin T levels. EAT thickness also correlated significantly with high SYNTAX scores. However, it did not correlate with GRACE score [8]. The study of Park EM et al., which evaluated the role of EAT thickness on the short

term prognostic role in ACS patients, the mean EAT was found to be 5.36 mm. Multivariate analyses demonstrated that EAT could be used as an independent predictive factor of the acute cardiac events within 30 days [56].

Other modalities like MDCT and MRI for estimating Epicardial fat have also extensively compared EAT and the ability to predict significant CAD. Gorter et al., observed that the EAT was significantly higher in patients with mutivessel disease than those without CAD (100 Vs 67 cm^3), even in patients with low BMI. The study also noted that the EAT volume (108 Vs 69 cm^3) and the PAT thickness (10 Vs 8.2mm) was increased in subjects with high CAC score than in patients with minimal or normal CAC score [57]. Christopher L. Schlett et al., analysed PAT volume by CT and the CAD by CT angiography in patients admitted in emergency room with acute chest pain. It was observed that compared to patients without high risk lesions and normal coronaries, the patients with high risk lesions had significantly higher PAT. The association of PAT volume and the high risk coronary lesions were independent of patients clinical characteristics and obesity parameters [58]. Other studies using MDCT also demonstrated the same findings: EAT volume correlated with the components of Met S (waist circumference, BMI, fasting serum glucose, HDL-cholesterol, total cholesterol and triglycerides levels), coronary lesions, Gensini score, and coronary calcium scores [59, 60].

Cardiac Magnetic resonance has also been used to study the relation between EAT and CAD. In the study of Kim et al., diabetic patients with no clinical evidence of CAD underwent CMR for evaluation of epicardial fat and the coronary vessels were evaluated using MR angiography. They found that EATT was significantly higher in patients with coronary artery stenosis (13.0 ± 2.6 mm vs. 11.5 ± 2.1 mm) than in those without. They also noted that EATT was shown to independently correlate with significant CAD even after adjusting for risk factors [61].

The ability to predict future CAD was evaluated in the MESA study. In this study 980 patients were followed up for an average period of 5 years. Base line CT was done to assess the coronaries and also calculate the pericardial fat. It was found that the waist circumference and the volume of pericardial fat was higher in patients with CAD and also the participants with higher quartiles had more than double the risk of coronary artery disease [62].

Although the relation between EAT and CAD has been analysed in various studies, there are some studies which found no correlation between the two and some studies which showed only a weak correlation. Chaowalit et al., did not find any significant association between the EATT and the CAD severity or any of the clinical variables [63]. Even in studies which showed a positive relation to the total EAT volume

and also to the atherosclerotic plaques or coronary artery calcium, paradoxically it was found that the EAT volume was not related in a dose dependent manner to the coronary atherosclerosis severity or with the coronary calcium score [52, 57, 64]. The MESA study also showed that this correlation was only marginally significant after including waist circumference in the analysis. In post menopausal women it was found that the positive correlation between the coronary artery calcium score and pericoronary epicardial fat thickness was not significant after it was adjusted for waist circumference [47].

EAT and association with other vascular diseases:

Arterial stiffness is a important feature of vascular aging and it is considered an risk factor for cardiovascular disease. In the MESA study the association between pericardial fat and carotid stiffness was determined in 5,770 participants. Pericardial fat was measured by CT scan. The study showed that elevated pericardial fat is linked with higher carotid stiffness. This association was independent of traditional risk factors and obesity [65]. In a small study the endothelial function by flow mediated vasodilatation was compared with epicardial fat thickness, which found no correlation between these two parameters [66].

EAT and its association with other diseases:

Non alcoholic fatty liver disease has been recently evaluated and it has been accepted to have a casual association with increased cardiovascular disease risk. A study which evaluated the association between EAT and CIMT found a statistically significant correlation between these two parameters and NAFLD [67].

Thoracic peri aortic fat and epicardial fat were evaluated by MDCT in ESRD patients undergoing peritoneal dialysis. The study demonstrated that peri aortic fat, epicardial fat, coronary artery calcification and thoracic artery calcification was significantly higher in the peritoneal dialysis group [68].

HIV infection has been associated with accelerated atherosclerosis and increased cardiovascular risk. HIV patients on HAART therapy develop a drug induced lipodystropy with metabolic syndrome. HIV-infected patients with metabolic syndrome and LDS linked to HAART showed greater EATT and IMT (8 Vs 6.5 mm; 0.71 Vs 0.66 mm, respectively) than HIV-infected subjects on HAART without LDS [69].

Vascular inflammatory diseases might have potential for endothelial dysfunction and early atherosclerosis. Based on this concept, FMD and EAT were evaluated in patients with Bechet's disease which is an autoimmune vasculitis disease. EAT thickness correlated with the

disease activity. The FMD was negatively correlated with disease activity, EAT thickness, gamma glutamyl transferase levels and age [70].

ASSESSMENT OF EPICARDIAL ADIPOSE TISSUE:

ECHOCARDIOGRAPHIC ASSESSMENT:

It is a non invasive and safe method. Echocardiography along with MDCT is considered one of the gold standards for assessment of Epicardial fat. Iacobellis et al, have shown that the thickness of epicardial fat in the free wall of right ventricle assessed by echocardiography, correlated significantly with the visceral adipose tissue determined by MRI [6]. Most of the studies of EAT measurement by echocardiography had found good reproducibility and reliability [5, 6, 9, 53, 54, 55]. EAT measurement by echocardiography is done by measuring the echo free space anterior to free wall of right ventricle. If the Epicardial tissue is massive it resembles a hyper-echoic space. It is important to make certain that EATT is not measured at an angle since it leads to incorrect measurements [72]. Parasternal long axis view is used in most of the studies for measurement of Epicardial fat. It is seen as an echo free space anterior to the RV free wall and is measured during end diastole [5, 6, 53]. This method is adopted with some variations in most of the studies. Some studies have used combination of parasternal long axis and parasternal short axis

measurements, while in the study by Toufan M et al., the EATT was measured at parasternal long axis view at anterior aspects proximal and distal to RV outlet tract (RVOT), and modified long axis view used to measure of RV apical fat pad thickness. Three measurements are usually taken and averaged since there is some variation in the longitudinal thickness of epicardial fat.

A few limitations exist with this method. In some patients the echocardiographic window and the pictures may not be clear, which causes difficulty in estimating the EAT thickness. The other problem is that in patients with pericardial fluid, it may be confused with epicardial fat, especially if the fluid is localized.

COMPUTED TOMOGRAPHY:

CT is often considered the gold standard in imaging EAT, because among the three imaging modalities (echocardiography, magnetic resonance imaging, and MDCT), MDCT offers a novel opportunity to assess in vivo distribution of EAT in high-resolution three-dimensional views. Echocardiogram is dependent on acoustic windows and cannot provide an adequate window to assess all cardiac segments, whereas magnetic resonance imaging has limitations in spatial resolution in the through-plane dimension [40]. As discussed before studies using both MDCT and Dual slice CT have shown that EAT volume calculated with CT scan

correlated with the indices of metabolic syndrome (BMI, waist circumference, hypertension and impaired fasting glucose) [35], CAC score and to the severity of CAD [57, 58, 60].

EAT is measured by multidetector computed tomography (MDCT) in three dimensions: regional thickness, cross-sectional areas, and total volume. The measurement is made sequentially first by measuring the regional thickness by obtaining multiplane reconstructions of the MDCT data in the standardized ventricular short-axis planes at the apical, mid-cavity, and basal levels and also in the horizontal long-axis plane. Next step is to measure the cross sectional areas. The cross sectional areas are measured in all MDCT planes. To obtain EAT cross-sectional areas, measurements of thin-slab volume of EAT from two contiguous images are made by tracing the pericardium. All the axial images are loaded into a workstation and the pericardium is manually traced in these images, which will be used to calculate the entire EAT volume [40]. The EAT volume in studies ranged from 107± 43 (Wang et al (36)) to 110 ± 44 (Gorter et al [57]).

There are few issues with using CT for measuring EAT. First being the ionising radiation, which posses a hazard for the patient. Secondly the pericardium is not clearly visualized in the CT, which is important to delineate epicardial from paracardial fat, especially in lean individuals [72].

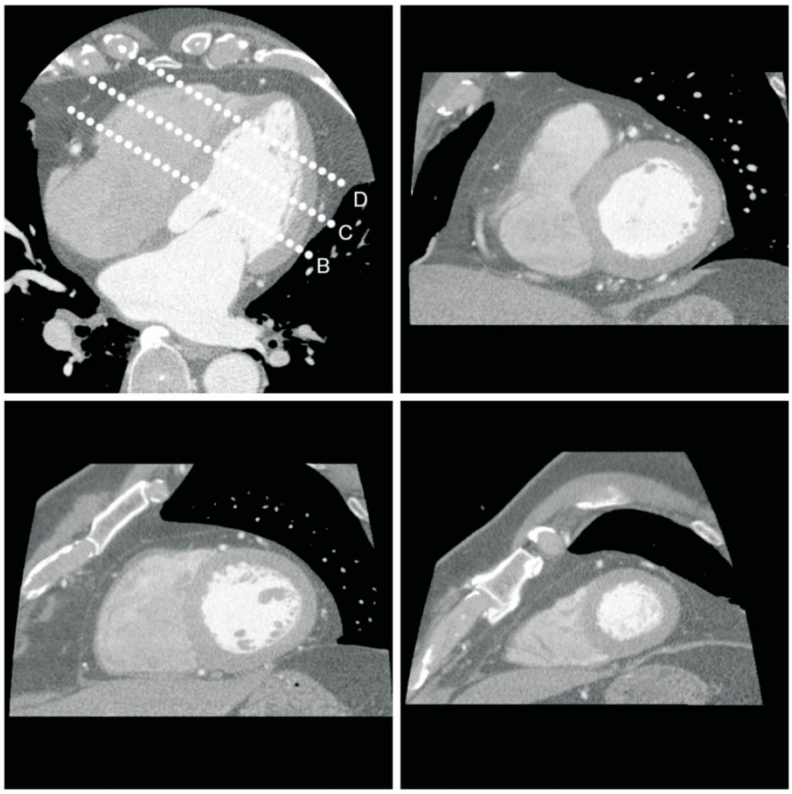


FIGURE 3: MDCT reconstructed imaged of heart showing EAT

CARDIAC MAGNETIC RESONANCE

Recently trials have shown that EAT as assessed by CMR in obese individuals and in metabolic syndrome significantly correlated with inflammatory mediators and vascular function [73, 74]. It was also effectively used to assess the correlation with EAT by CMR and the severity of CAD as calculated by MR angiography in asymptomatic type 2 diabetics [61]. Also as discussed earlier, the 3.0 T MRI available now, has demonstrated that it was able to delineate and quantify visceral fat as good as CT and better than MRI with lesser tesla.

CMRI acquisition requires a standardized protocol. Gating is done using a cardiac coil and electrocardiographic triggering. Patients hold their breath for 10 to12 seconds during the acquisition period. Cardiac adipose tissue scans are obtained by fast spin-echo T1-weighted sequences with oblique axial orientation, to correctly study the horizontal long axis of the heart. EPI was defined as any adipose tissue located within the pericardial sac. Fat thickness in the left atrioventricular groove was measured. The EAT is calculated at the end-diastolic phase in the horizontal long-axis plane in cine MRI. Maximal EAT thickness is estimated as the distance from the myocardial surface to the pericardium [61, 73].

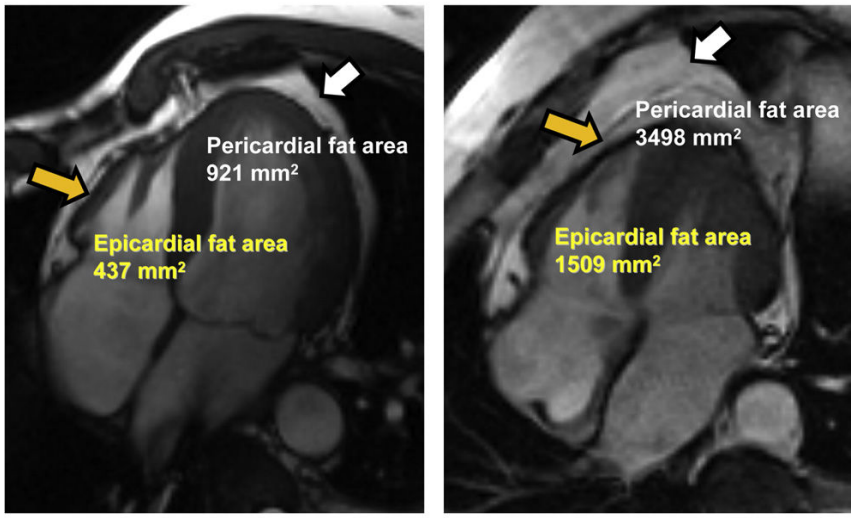


FIGURE 4: MR image showing normal and increased epicardial fat.

EAT assessment in Indian population:

There is only one study undertaken in Indian population undertaken by Ranjan Shetty et al., which found that the mean epicardial fat is 2.6 ± 1.3 mm (range 0.3 to 7.0 mm). Like previous studies it also found that epicardial fat correlated with waist circumference and age. In this study the epicardial fat also correlated with BMI, unlike most of the other studies [75].

CAROTID INTIMA MEDIA THICKNESS

Gradual increase in thickness of the intima and media layers is one of the first clinically quantifiable aspects of atherosclerosis [16, 17]. Carotid intima media thickness (CIMT) uses ultrasound to assess the arterial wall thickness of the carotid artery. CIMT is defined as the combination of thickness of intima and the media layers, and is measured as the distance that spans the intima- luminal and the medial- adventitial interfaces. CIMT is an excellent validated tool for assessment of cardiovascular risk. Increased CIMT has shown to correlate with known cardiac risk factors, the ability to quantify the severity of atherosclerosis and also serves as a marker of subclinical cardiovascular risk assessment [13, 76].

Normal and abnormal CIMT:

CIMT values vary within the healthy population according to age, gender, and race. The normal and abnormal values have been described based on this distribution [77]. It has been demonstrated from large population studies that CIMT increases with age [14], is greater in men than women [13]. It has also been shown to vary according to race, with highest range in African Americans, intermediate in Caucasians and least in Hispanics [77]. The normal CIMT is often considered to be the ≤ 75% percentile of CIMT distribution, a value which generally corresponds to an absolute value of CIMT less than 1.0mm. Focal plaque is defined as a focal increase in CIMT > 1.5 times that of surrounding CIMT.

Studies on Indian population has shown that mean CIMT is lower with average CIMT in patients with CAD, stroke and in diabetics was about 0.8mm [78, 79, 80]. Studies have used a cut off of 0.8 mm to define a significant increase in CIMT [79].

Prognostic impact of CIMT:

CIMT has been shown to be clearly associated with previous exposure to smoking, elevated LDL and hypertension [81]. The cardiovascular Health Study (CHS) and the ARIC study have clearly demonstrated a relationship between increased CIMT and CAD, and its ability to predict future cardiovascular risk [14, 82]. Baldassarre et al.,

explored whether CIMT and Framingham risk score could be combined to improve the ability to predict Cardio vascular events in low to intermediate risk dyslipidemic patients. He concluded that both CIMT and FRS were independent predictors of CV events [83]. Stein et al. evaluated the effect of age on the calculated CIMT determined by vascular age. They reclassified the cardiovascular risk according to FRS in patients without known CAD. The study showed that when "vascular age" is substituted for chronological age the Framingham 10-year risk score increased from 6.5 to 8 percent [84].

CIMT and relation to severity of CAD:

Kablak et al explored the correlation between CIMT and the severity of CAD in patients undergoing coronary angiography [85]. The study found a significant correlation between the advancing severity of CAD and the mean CIMT. It also showed that with mean CIMT more than 1.15 mm, the probability of having CAD was 94%, with specificity of 80% and sensitivity of 65% in the patients with a high risk of CAD.

ASSOCIATION BETWEEN EAT THICKNESS AND CIMT

The relationship between these two parameters has been done in various groups of patients. In a study of Cetin et al. in diabetic patients, they found that diabetic patients had significant higher CIMT and EAT when compared to controls (0.76 ± 0.17mm versus 0.57 ± 0.14 mm, $P < 0.001$ and 6.0 ± 1.5mm versus 4.42 ± 1.0 mm, $P < 0.001$,

respectively) [86]. In a small study of 40 patients with metabolic syndrome, the EAT and CIMT were significantly increased in patients with MetS compared to controls (7.2 ± 2 mm vs. 5.7 ± 1.9 mm; P = 0.001, 0.74 ± 0.1 mm vs. 0.59 ± 0.1 mm; P < 0.01, respectively) [87]. Similar results were demonstrated in a study, but this involved a very young group of study population with metabolic syndrome [88]. EAT and CIMT have also shown positive correlation in varied groups like HIV patients on HAART therapy and in patients with NAFLD [89, 90].

EFFECT OF THERAPY ON EPICARDIAL FAT:

Kim MK et al. evaluated the effect of exercise on epicardial fat in obese men [91]. The pre and post exercise epicardial fat and abdominal visceral fat were evaluated with CT scan. After a supervised exercise program for 12 weeks it was found that the epicardial fat had significantly reduced. Also it was shown that the change in epicardial fat was twice when compared to change in waist circumference, BMI and body weight of original values. The change in systolic blood pressure, and change in quantitative insulin sensitivity check index were independently related to the change epicardial fat thickness. However in a small study of 12 diabetics on moderate intensity exercise of 6 months there were significant

reductions in visceral fat, subcutaneous fat and fasting triglyceride level without any significant change in epicardial fat [92].

Park et al. studied the effect of statins on the epicardial adipose tissue in patients undergoing PCI and were followed up for months. It was found that atorvastatin at a dose of 20mg had a significant effect on EAT when compared to simvastatin and ezetimibe combination. The difference may be related to the different statin doses used, however this study clearly showed a positive effect of statin on epicardial fat [93].

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MATERIALS AND METHODS

Study Design: Retrospective case control study

Duration: August 2013 to January 2013

Setting: Govt. Stanley Medical College and Hospital, Chennai.

Study Group: Patients undergoing coronary angiogram in the department of cardiology were enrolled for the study. Of the total 340 patients who underwent angiogram during this period, after 90 patients were excluded as per criteria, 250 patients were included for the study. Enrolled patients were divided into two groups for the study.

i. Group with abnormal Coronary angiogram
ii. Control group with normal coronary angiogram.

Inclusion criteria: Patients aged from 18 to 75 years who are referred for diagnostic coronary angiogram for indications including:

Chronic stable angina,

Unstable angina and NSTEMI,

Acute and Recent STEMI.

Exclusion criteria

Age less than 18 and age more than 75 years,

Poor Echo Window,

Pericardial effusion,

Routine CAG in patients undergoing Valve replacement,

CKD patients undergoing routine CAG before renal transplantation.

Data collection technique and tools

Ethical issues:

As this study involves the taking of blood investigations, and an invasive diagnostic procedure such as coronary angiogram, all patients and their relatives were explained the study design at the time of enrolment.

Informed consent was obtained from all patients at the time of admission (a copy is enclosed).

The following demographic and clinical data were obtained as soon as the patient was admitted and enrolled.

A proforma (a copy enclosed) for each patient was filled which included the following:

- Age of the patient
- Symptom history

- A complete risk factor profile (including diabetes, hypertension, dyslipidemia, smoking, family history, prior history of CAD).
- physical examination
  - Including the pulse rate, blood pressure, cardiac & respiratory system clinical examination.
  - Height and body weight - used to calculate body mass index.
  - Minimum waist circumference (in centimetres) (minimum circumference between the lower rib margin and the iliac crest, mid waist) and maximum hip circumference (in centimetres) (the widest diameter over the greater trochanters) are measured while the subjects are standing with their heels together.
    - We used waist circumference (88 cm in women and 102 cm in men) as the threshold of predominant truncal/ abdominal fat distribution.

Investigations

- Complete blood investigations including blood counts, blood sugar, urea and creatinine, liver function test, HBsAg, HCV, and HIV were taken at admission.
- Lipid profile was done in overnight fasting state
- A 12 lead electrocardiogram was obtained. ECG using right sided leads or posterior leads was taken if necessary.

- Echocardiogram was performed at time of admission and one day after coronary angiogram (to calculate EAT thickness).
- e GFR was calculated by the Cockroft Gault formula
- Chest x ray PA view
- Coronary angiogram was performed during index hospitalization.

SAMPLE COLLECTION

Echocardiographic measurement

All patients underwent echocardiography day after coronary angiogram for assessment of EAT. Echocardiography was performed by experienced cardiologists using Philips HD 11 XE ultrasound machine (Koninklijke Philips NV, Netherlands). The images are stored in the computerised database for further calculations. The offline measurement of EAT was done by 2 cardiologists who were blinded to the clinical and angiographic data. With the patient in left lateral position, the EAT thickness is measured from the free wall of RV in parasternal long axis view. Epicardial fat is identified as an echo-free space between the Right ventricular myocardium and the visceral pericardium (epicardium), measured perpendicularly on the free wall of right ventricle at end-diastole for 3 cardiac cycles [5, 6, 53]. To standardise the measuring point the aortic annulus is used as the anatomic landmark. The measurements were performed at a point on the free wall of the RV, along the midline of the

ultrasound beam and perpendicular to the aortic annulus. The average values from 3 cardiac cycles were used for statistical analysis.

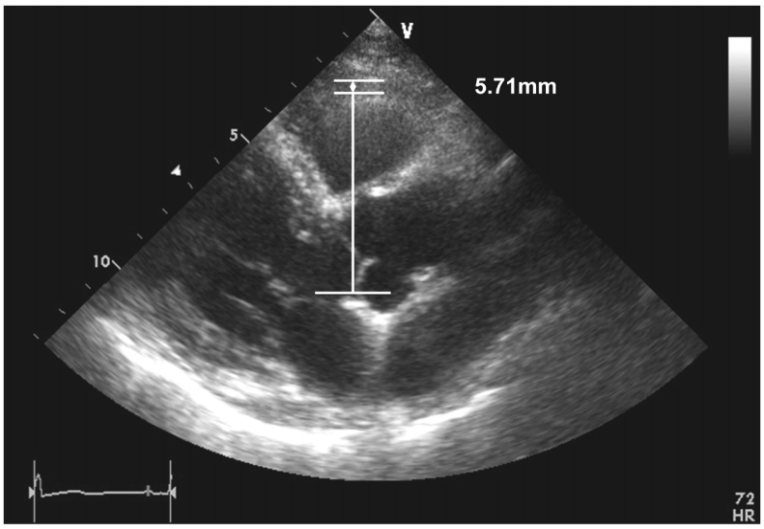


FIGURE 5: Example of estimation of EAT thickness by TTE.

Ultrasound quantification of Carotid intima media thickness

Carotid arteries were evaluated using Esoate my lab50 ultrasound machine (Genoa, Italy). Measurements were done with a 10 MHz transducer. All examinations were done by experienced radiologist who were unaware of the clinical, EAT or the angiographic data. Examination involved scanning of involved a transverse and longitudinal scanning of the common carotid artery, bifurcation, and internal carotid artery. The CIMT was measured on the far wall at 1 cm from bifurcation of the common carotid artery as the distance between the lumen intima interface and the media-adventitia interface. At least three measurements

were performed on both sides, and the average measurement was taken as the CIMT [85, 86, 94]. All measurements were made at a plaque-free site. The IMT was assessed as normal if it did not exceed 1 mm. Plaque was defined as a focal thickening of the intima-media complex greater than 1.3 mm [94].

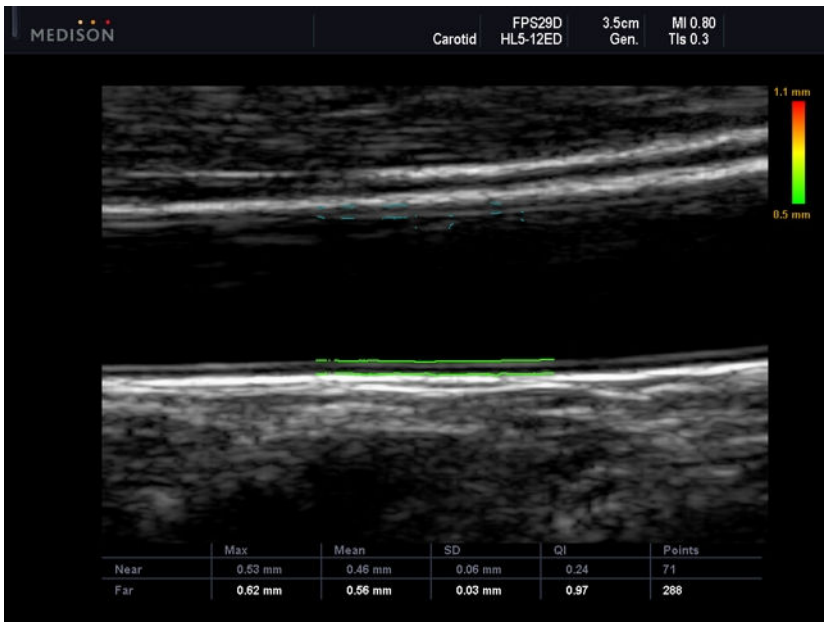


FIGURE 6: Example of estimation of carotid intima media thickness

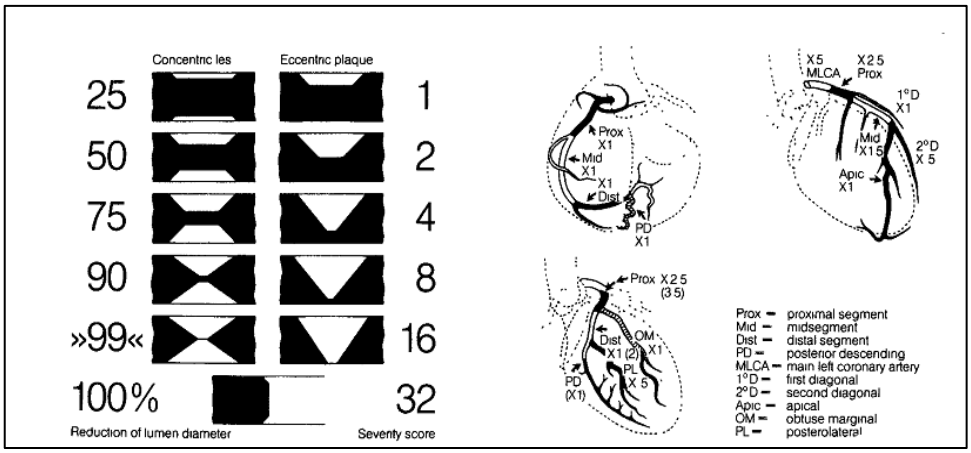
Coronary Angiography

Coronary angiography is performed in with Seimens axiom altis and images recorded at a frame rate of 15/s. The severity of coronary atherosclerotic lesions is evaluated from at least 3 angiographic projections. Significant stenosis is defined as a diameter stenosis of 50% or greater.

The Gensini score was used to calculate the severity of angiographic lesions and verified by a team of experts [95]. This score

assigns heavier weight to more severe luminal narrowing and also gives weight to the segment of coronary artery involved according to vessel size and importance; segments that serve larger regions are more heavily weighted.

A 25% diameter reduction is given a score of 1. Likewise diameter reduction is scored as following; 50% reduction as 2; a 75% reduction as 4; a 90% reduction as 8; a 99% reduction is scored as 16 and 100% cut off is scored as 32. This score is multiplied by the weight given to coronary segments such as Left main 5; proximal LAD is given 2.5; mid LAD 1.5; distal LAD 1;the first diagonal is given 1 and second diagonal is given 0.5; proximal LCX is given 2.5 if nondominant and 3.5 if dominant; distal LCX is given 1 (nondominant) and 2 (dominant). OM s 1; PLB 0.5; PDA 1; RCA 1, each for proximal, middle and distal. The diameter reduction score is multiplied by the coronary segment score for each lesion. The total gives the Gensini score for the patient.



STASTICAL ANALYSIS

All statistical analysis was done using SPSS statistical software (SPSS version 17.0 for windows, Inc., Chicago, IL, USA). The data are expressed as mean for quantitative data. The descriptive data are expressed as frequency and percentage. The differences between the groups were analyzed using student *t*- test for continuous variables and Chi- square test for discrete variables. One-way ANOVA was used for groups with more than two means. Correlation analysis was performed using Pearson correlation. Linear regression was used to assess the independent determinants of EAT thickness, LV mass, CIMT and GENSINI score. Logistic regression was used to assess the independent determinants for coronary artery disease. Level of statistical significance is set a P value < 0.05.

RESULTS

The overall mean age of the patients was 53.05 ± 9.76. 77.9% were males. Among the risk factor prevalence 42.6 % were diabetics, 48.4% had hypertension and 38.9% were smokers.

Of the patients undergoing coronary angiogram 28.7% had chronic stable angina, 26.2% had UA/NSTEMI and 45.1% had myocardial infarction. A total of 79 patients had normal or insignificant CAD (32.3%). Single vessel disease was present in 44 patients (18%) and double and triple vessel disease was present in 61 patients (25%) in each group. The mean GENSINI score was 27.82 ± 28.92.

The overall mean EAT thickness was 4.04 ± 1.94mm (maximum of 8.1mm). The mean CIMT was 0.84 ± 0.21mm.

Distribution of EAT thickness

EAT thickness	Frequency	Percent
less than 3	73	29.9
3 to 4	45	18.4
4 to 6	82	33.6
more than 6	44	18.0
Total	244	100.0

Percentiles	EAT thickness
25	2.425
50	4.200
75	5.400

Study population characteristics according to EAT thickness.

TABLE 1: **EAT AND SEX**

	SEX	N	Mean	Std. Deviation	Std. Error Mean	t = 2.226, df = 242, Sig. (2-tailed) P = 0.034.
EAT	male	190	4.196	1.9027	.1380	
	female	54	3.533	2.0216	.2751	

The association between EAT thickness and the sex of the patient was statistically significant at a P value of 0.034.

TABLE 2: **EAT AND DIABETES**

	DM	N	Mean	Std. Deviation	Std. Error Mean	t = 3.320 df = 242 Sig. (2-tailed) P < 0.001
EAT	yes	104	4.519	1.9062	.1869	
	No	140	3.700	1.9061	.1611	

The diabetes group had a significantly higher mean EAT compared to non diabetic group with a P value of 0.001.

TABLE 3: **EAT AND AGE**

AGE	Mean	N	Std. Deviation
less than 30	3.573	26	1.9272
40 to 60	3.991	159	1.8478
more than 60	4.415	59	2.1683
Total	4.049	244	1.9450

ANOVA Table

		Sum of Squares	df	Mean Square	F	Sig.
EAT * AGE	Between Groups (Combined)	14.336	2	7.168	1.909	.150
	Within Groups	904.969	241	3.755		
	Total	919.305	243			

Measures of Association

	Eta	Eta Squared
EAT * AGE	.125	.016

The association between various age groups and difference in EAT thickness was not statistically significant P=0.15.

TABLE 4: **EAT AND HYPERTENSION**

	HTN	N	Mean	Std. Deviation	Std. Error Mean	
EAT	Yes	118	4.109	1.8895	.1739	t = 0.466 df = 242 Sig.(2-tailed) P = 0.641
	No	126	3.993	2.0015	.1783	

EAT was not significantly different between hypertensives and normotensives (P=0.641).

TABLE 5: **EAT AND SMOKING**

	smoking	N	Mean	Std. Deviation	Std. Error Mean	
EAT	Yes	95	3.963	2.0875	.2142	t = -0.542 df = 242 Sig. (2tailed) P = 0.584
	No	149	4.104	1.8537	.1519	

EAT was not statistically different between smokers and non smokers P=0.584

TABLE 6: **EAT AND BMI**

		EAT thickness					Total
		less than 3	3 to 4	4 to 6	more than 6	Mean ± SD	
BMI	less than 25	71	42	63	31	3.82 ± 1.91	207
	25 to 30	2	3	16	11	5.25 ± 1.67	32
	More than 30	0	0	3	2	5.87 ± 0.89	5
Total		73	45	82	44		244

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)
Pearson Chi-Square	22.913[a]	6	.001
Likelihood Ratio	26.666	6	.000
Linear-by-Linear Association	20.585	1	.000
N of Valid Cases	244		

a. 4 cells (33.3%) have expected count less than 5. The minimum expected count is .90.

At Chi-square =22.9 and degree of freedom of 6 the EAT thickness was significantly different among the groups of BMI, P=0.001. The Pearson's R value is 0.291 indicating a weak correlation between BMI and EAT thickness.

EAT AND WAIST CIRCUMFERENCE

TABLE6: Waist circumference cut off set at 85 cm for males and 80 cm for females

		EAT THICKNESS				Mean ± SD	Total
		less than 3	3 to 4	4 to 6	more than 6		
WC	Normal	58	20	16	4	2.74 ± 1.72	98
	increased	15	25	64	42	4.92 ± 1.55	146
Total		73	45	80	46		244
Pearson Chi-square=79.718, df=3, Asymp. Sig(2-sided) P < 0.001. Pearson's R value = 0.559.							

TABLE 8: Waist circumference cut off set at 102 cm for males and 88 cm for females

		EAT THICKNESS				Mean ± SD	Total
		less than 3	3 to 4	4 to 6	more than 6		
WC	Normal	72	45	66	41	3.94 ± 1.96	224
	increased	1	0	14	5	5.19 ± 1.20	20
Total		73	45	80	46		244
Chi-square=18.17, df=3, Asymp. Sig.(2-tailed) P <0.001 Pearson's R value = 0.202							

EAT is significantly associated with waist circumference at both cut off points, P<0.001. However the correlation was good with cut off of 85 for males and 80 for females (r = 0.559) and weak with waist circumference cut off set at 108 for males and 88 for females (r = 0.202).

EAT AND BLOOD PRESSURE:

TABLE 9: EAT AND SYSTOLIC BLOOD PRESSURE

		EAT 2					Total	
		less than 3	3 to 4	4 to 6	more than 6	Means ± SD		
SBP	less than 140	61	27	57	24	3.75 ± 1.95	169	Chi-square = 15.82, df = 3, P = 0.002
	More than 140	12	18	23	22	4.71 ± 1.75	75	
Total		73	45	80	46		244	

Chi-square = 15.82 with 3 degrees of freedom. Systolic BP correlated with EAT thickness, P = 0.002.

TABLE 10: EAT AND DIASTOLIC BLOOD PRESSURE

		EAT 2					Total	
		less than 3	3 to 4	4 to 6	more than 6	Mean ± SD		
DBP	less than 90	69	31	60	35	3.89 ± 2.01	195	Chi-square = 14.73, df = 3, P = 0.002
	more than 90	4	14	20	11	4.67 ± 1.47	49	
Total		73	45	80	46		244	

Chi-square = 14.73 with 3 degrees of freedom. Diastolic BP correlated with EAT thickness, P = 0.002.

BLOOD SUGAR AND EAT THICKNESS

TABLE 11:

	RBS	N	Mean	Std. Deviation	Std. Error Mean	
EAT	Less than 140	186	3.978	1.9192	.1407	t = -0.918, df = 242, Sig. (2-tailed) P = 0.36
	More than 140	58	4.256	2.0203	.2653	

TABLE 12

		EAT	RBS
EAT	Pearson Correlation	1	.116
	Sig. (2-tailed)		.069
	N	244	244
RBS	Pearson Correlation	.116	1
	Sig. (2-tailed)	.069	
	N	244	244

**Correlation is significant at the 0.01 level (2-tailed).

Random blood sugar value with a value set at 140 mg/dl did not significantly different EAT thickness between the two groups (P = 0.36). Pearson correlation between RBS was also weak (r = 0.116) and not statistically significant (P = 0.069).

LIPID PROFILE AND EAT THICKNESS:

TABLE 13: Total Cholesterol and EATT

	T. CHL	N	EAT Mean ± SD	t = -1.844, df = 242, P = 0.06
EAT	Less than 200	166	3.893 ± 1.88	
	More than 200	78	4.382 ± 2.04	

TABLE 14: HDL- C and EATT

	HDL	N	EAT Mean ± SD	t = 3.31, df = 242, P = 0.001
EAT	Low	85	4.605 ± 1.91	
	Normal	159	3.752 ± 1.90	

TABLE 15: LDL-C and EATT

LDL	N	EAT Mean	Std. Deviation	F = 0.995, df = 3, P = 0.41
< 100	110	3.831	1.9671	
100 – 130	68	4.131	1.8771	
130 – 160	13	4.385	2.0952	
>160	53	4.314	1.9510	
Total	244	4.049	1.9450	

TABLE 16: Triglyceride level and EATT

TGL	N	EAT Mean	Std. Deviation	F = 5.4, df = 2, P = 0.005
<150	74	3.658	1.8640	
150 – 200	105	3.926	1.9714	
>200	65	4.693	1.8610	
Total	244	4.049	1.9450	

TABLE 17: TC/HDL ratio and EATT

TC/HDL	N	EAT Mean	Std. Deviation	F = 5.08, df = 2, P = 0.007
< 3.5	68	3.491	1.9181	
3.5 – 5	109	4.099	1.8031	
>5.0	67	4.535	2.0760	
Total	244	4.049	1.9450	

The total cholesterol and HDL cholesterol groups were compared using student t-test. The EATT was not statistically different in the cholesterol group (P = 0.06). EATT was statistically different among the HDL normal and low group (P = 0.001). TGL, LDL and the TC/HDL ratio was analyzed using ANOVA. EAT was not statistically significant among the various LDL levels (P=0.41). The TGL (P = 0.005) and TC/HDL ratio (P = 0.007) had significantly different EAT thickness among the sub groups.

EAT AND LV MASS

TABLE 18:

		LV MASS		Total	LV Mass Mean ± SD	Chi square = 10.79, df = 3, P = 0.013.
		Normal	Increased			
EAT	<3mm	48	26	74	190.61± 55.019	
	3-4mm	26	20	46	211.96 ± 70.108	
	4-6mm	38	44	82	234.20 ± 79.178	
	>6mm	15	27	42	257.98 ± 84.716	
Total		127	117	244		
EAT Mean ± SD		3.62 ± 1.88	4.50 ± 1.90			F = 0.002, df =1, t = -3.610 Sig. P < 0.001

TABLE 19: Diastolic Dysfunction and EATT

		EAT				Total	Chi –square = 83.2, df=9, P<0.001
		Less than 3mm	3 – 4mm	4 – 6mm	More than 6mm		
Diastolic Dysfunction	Grade 0	48	11	14	2	75	
	Grade 1	24	21	41	16	102	
	Grade 2	2	12	25	26	65	
	Grade 3	0	1	0	1	2	
Total		74	45	80	45	244	

LV mass was significantly different in the various EATT groups and also EATT was significantly different between grades of diastolic dysfunction.

EAT THICKNESS AND CORONARY ARTERY DISEASE

TABLE 20: EAT thickness cutoff at 3 mm.

			Coronary artery disease		Total	
			No	yes		
EAT thickness	less than 3 mm	Count	59	15	74	Chi-square=108.77 df = 1, P<0.001
		% within EAT thickness	**79.7%**	20.3%	100.0%	
		% within Coronary artery disease	**74.7%**	9.1%	30.3%	
	More than 3 mm	Count	20	150	170	
		% within EAT thickness	11.8%	**88.2%**	100.0%	
		% within Coronary artery disease	25.3%	**90.9%**	69.7%	
Total		Count	79	165	244	
		% within EAT thickness	32.4%	67.6%	100.0%	
		% within Coronary artery disease	100.0%	100.0%	100.0%	

TABLE 21:

		EAT thickness > 3	Coronary artery disease
EAT thickness > 3	Pearson Correlation	1	.657(**)
	Sig. (2-tailed)		.000
Coronary artery disease	Pearson Correlation	.657(**)	1
	Sig. (2-tailed)	.000	

**Correlation is significant at the 0.01 level (2-tailed).

CAD significantly correlated with a EAT thickness cut off set at 3mm, P<0.001. The correlation between EAT thickness and CAD was also good r=0.657. Sensitivity was 90.9% and specificity was 74.7%.

TABLE 22: EAT thickness cut off at 4mm

			Coronary artery disease		Total	
			No	yes		
EAT thickness	less than 4 mm	Count	73	47	120	Chi-square= 87.332, dt = 1, P<0.001
		% within EAT thickness	**60.8%**	39.2%	100.0%	
		% within Coronary artery disease	**92.4%**	28.5%	49.2%	
	More than 4 mm	Count	6	118	124	
		% within EAT thickness	4.8%	**95.2%**	100.0%	
		% within Coronary artery disease	7.6%	**71.5%**	50.8%	
Total		Count	79	165	244	
		% within EAT thickness	32.4%	67.6%	100.0%	
		% within Coronary artery disease	100.0 %	100.0 %	100.0%	

TABLE 23:

		Coronary artery disease	EAT thickness > 4
Coronary artery disease	Pearson Correlation	1	.575(**)
	Sig. (2-tailed)		.000
EAT thickness > 4	Pearson Correlation	.575(**)	1
	Sig. (2-tailed)	.000	

**Correlation is significant at the 0.01 level (2-tailed).

CAD significantly correlated with a EAT thickness cut off set at 4mm, P<0.001. The correlation between EAT thickness and CAD was average, r=0.575. Sensitivity was 71.5% and specificity is 92.4%.

TABLE 24: EAT thickness cut off at 6 mm.

			Coronary artery disease		Total	
			No	yes		
EAT thickness	less than 6 mm	Count	77	123	200	Chi-square=18.91, df = 1 P<0.001.
		% within EAT thickness	**38.5%**	61.5%	100.0%	
		% within Coronary artery disease	**97.5%**	74.5%	82.0%	
	more than 6 mm	Count	2	42	44	
		% within EAT thickness	4.5%	**95.5%**	100.0%	
		% within Coronary artery disease	2.5%	**25.5%**	18.0%	
Total		Count	79	165	244	
		% within EAT thickness	32.4%	67.6%	100.0%	
		% within Coronary artery disease	100.0 %	100.0 %	100.0%	

TABLE 25:

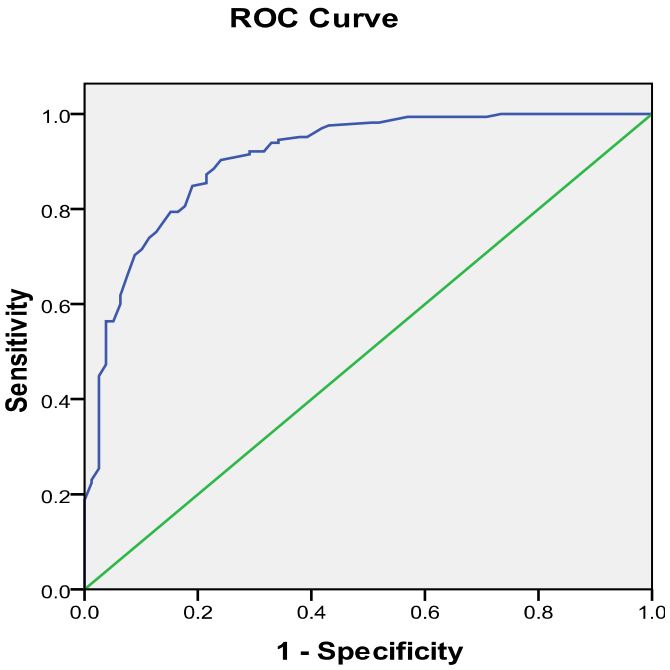
		Coronary artery disease	EAT thickness > 6
Coronary artery disease	Pearson Correlation	1	.279(**)
	Sig. (2-tailed)		.000
EAT thickness > 6	Pearson Correlation	.279(**)	1
	Sig. (2-tailed)	.000	

**Correlation is significant at the 0.01 level (2-tailed).

CAD correlated significantly with a EAT thickness cut off set at 6mm, P<0.001. The correlation between EAT thickness and CAD was poor, r=0.279. Sensitivity was 25.5% and specificity was 97.5%.

Receiver operating characteristics (ROC) curve:

To determine the high risk value Of EATT to predict CAD.



Diagonal segments are produced by ties.

Area Under the Curve

Test Result Variable(s):EAT

Area
.909

Receiver operating characteristics (ROC) curve which shows sensitivity and specify of the EAT thickness to determine cut off high risk value. The area under the ROC curve is 0.909 which indicates a very good accuracy of the test. The coordinates of the curve showed that a value of **3.45mm** produced the best combination of sensitivity (85%) and specificity (78.5%).

CIMT and correlation with CAD

TABLE 26: CIMT and CAD

			CAD		Total	
			No	Yes		
CIMT	less than 1 mm	Count	60	21	81	Chi square = 26.47, df = 1, P < 0.001
		% within CIMT	**74.1%**	25.9%	100.0%	
		% within CAD	**75.9%**	12.7%	33.2%	
	more than or equal to 1 mm	Count	19	144	163	
		% within CIMT	11.7%	**88.3%**	100.0%	
		% within CAD	24.1%	**87.3%**	66.8%	
Total		Count	79	165	244	
		% within CIMT	32.4%	67.6%	100.0%	
		% within CAD	100.0%	100.0%	100.0%	

TABLE 27:

		CIMT	CAD
CIMT	Pearson Correlation	1	.628
	Sig. (2-tailed)		.000
	N	244	244
CAD	Pearson Correlation	.628	1
	Sig. (2-tailed)	.000	
	N	244	244

**Correlation is significant at the 0.01 level (2-tailed).

CAD correlated significantly with CIMT, P<0.001. The correlation was fair r=0.628. The sensitivity was 87.3 %, specificity was 75.9 %. Negative predictive value was 91.7%.

CAD and correlation with combined EATT and CIMT test.

TABLE 28: CAD and combined test of EATT (>3.4mm) and CIMT (>1mm)

Combined * CAD Cross tabulation						
			CAD			
			No	yes	Total	
Combined test	No	Count	58	13	71	**Chi square = 111.2, df = 1, P <0.001**
		% within Combined	**81.7%**	18.3%	100.0%	
		% within CAD	**73.4%**	7.9%	29.1%	
	Yes	Count	21	152	173	
		% within Combined	12.1%	87.9%	100.0%	
		% within CAD	26.6%	**92.1%**	70.9%	
Total		Count	79	165	244	
		% within Comb2	32.4%	67.6%	100.0%	
		% within CAD	100.0%	100.0%	100.0%	

TABLE 29:

		Combined test	CAD
Combined test	Pearson Correlation	1	.675**
	Sig. (2-tailed)		.000
	N	244	244
CAD	Pearson Correlation	.675**	1
	Sig. (2-tailed)	.000	
	N	244	244

CAD correlated significantly with the combined test of EATT and CIMT, P<0.001. The correlation was good r=0.675. The sensitivity was 92.1%, specificity was 73.4%. Negative predictive value was 81.7%

EATT and correlation with GENSINI score (CAD severity).

TABLE 30:

	EAT>3.4	N	Mean	Std. Deviation	Std. Error Mean	t = 9.431, df = 242, P <0.001.
GENSINI	No	84	7.17	14.482	1.580	
	Yes	160	38.66	28.728	2.271	

TABLE 31:

Correlations			
		GENSINI	EATT
GENSINI	Pearson Correlation	1	.660**
	Sig. (2-tailed)		.000
	N	244	244
EATT	Pearson Correlation	.660**	1
	Sig. (2-tailed)	.000	
	N	244	244
**Correlation is significant at the 0.01 level (2-tailed).			

GENSINI score correlated significantly with EATT, P<0.001. The correlation was good r=0.66. The sensitivity was 92 %, specificity was 66.7%. Negative predictive value was 87.7%.

CIMT and GENSINI Score

TABLE 32:

	CIMT	N	Mean	Std. Deviation	t = 8.11, df = 242, P<0.001.
GENSINI	Less than 1mm	81	6.56	12.382	
	More than 1mm	102	34.38	28.794	

TABLE 33:

Correlations			
		GENSINI	CIMT
GENSINI	Pearson Correlation	1	.519**
	Sig. (2-tailed)		.000
	N	244	244
CIMT	Pearson Correlation	.519**	1
	Sig. (2-tailed)	.000	
	N	244	244
**. Correlation is significant at the 0.01 level (2-tailed).			

GENSINI score correlated significantly with the CIMT, P<0.001. The correlation was average, r=0.519.

Combined EATT (>3.4mm) and CIMT (>1mm) test and correlation to GENSINI

TABLE 34:

	Combination test	N	Mean	Std. Deviation	t = 9.7, df = 242, P<0.001
GENSINI	yes	173	37.64	28.687	
	no	71	3.87	7.835	

TABLE 35:

Correlations			
		GENSINI	Combined test
GENSINI	Pearson Correlation	1	.531**
	Sig. (2-tailed)		.000
	N	244	244
Combined test	Pearson Correlation	.531**	1
	Sig. (2-tailed)	.000	
	N	244	244
**. Correlation is significant at the 0.01 level (2-tailed).			

GENSINI score correlated significantly with the Combined EATT and CIMT, P<0.001. The correlation was average, r=0.531.

TABLE 36: **MULTIPLE LINEAR REGRESSION FOR GENSINI SCORE:**

Model		Unstandardized Coefficients		Standardized Coefficients	t	Sig.
		B	Std. Error	Beta		
1	(Constant)	-.122	.532		-.230	.818
	AGE	.120	.070	.083	1.715	.088
	SEX	.196	.120	.098	1.627	.105
	DIAGNOSIS	-.026	.048	-.026	-.538	.591
	DM	-.169	.087	.101	-1.947	.043
	HTN	.001	.084	.001	.014	.989
	smoking	-.108	.098	-.064	-1.107	.269
	BMI	.253	.107	.131	2.359	.019
	WC	.230	.096	.136	2.399	.017
	WC2	-.068	.162	-.022	-.418	.676
	SBP	.145	.092	.081	1.570	.118
	DBP	-.064	.108	-.031	-.595	.552
	CIMT	.212	.070	.193	3.014	.003
	EATT	.227	.053	.303	4.323	.000
	combined	.298	.145	.154	2.060	.041
	T. CHL	.067	.180	.040	.371	.711
	LDL	.070	.072	.108	.969	.334
	HDL	-.112	.139	-.068	-.809	.420
	TGL	-.028	.076	-.025	-.364	.716
	TC/HDL	-.095	.120	-.086	-.795	.427

a. Dependent Variable: GENSINI

DM (P = 0.43), BMI (P=0.019), Waist circumference (P=0.017), EAT thickness (P<0.001) and CIMT (P=0.003) and Combined test (P=0.04) were independently predictive of GENSINI score. Other parameters are not independently related to GENSINI score.

TABLE 37: **MULTIPLE LOGISTIC REGRESSION ANALYSIS FOR SIGNIFICANT CORONARY ARTERY DISEASE**

Variables in the Equation

	B	S.E.	Wald	df	Sig.	Exp(B)	95% C.I.for EXP(B)	
							Lower	Upper
AGE	.126	.436	.084	1	.773	1.134	.483	2.667
SEX	.062	.734	.007	1	.932	1.064	.253	4.483
DIAGNOSIS	.146	.284	.265	1	.607	1.157	.663	2.019
DM	-.898	.549	2.681	1	.102	.407	.139	1.194
HTN	.712	.525	1.838	1	.175	2.039	.728	5.709
smoking	-.509	.629	.654	1	.419	.601	.175	2.062
BMI	.165	.680	.059	1	.808	1.180	.311	4.473
WC	2.033	.545	13.914	1	.000	7.638	2.624	22.232
SBP	.243	.563	.186	1	.666	1.275	.423	3.842
DBP	-.173	.646	.071	1	.789	.841	.237	2.987
T.CHL	-.792	1.113	.506	1	.477	.453	.051	4.011
LDL	1.005	.487	4.257	1	.039	2.732	1.052	7.096
HDL	-.590	.861	.470	1	.493	.554	.102	2.997
TGL	-.273	.461	.351	1	.554	.761	.308	1.879
TCHDL	-1.175	.778	2.283	1	.131	.309	.067	1.418
EAT >3.4	1.744	.499	12.221	1	.000	5.721	2.152	15.212
CIMT	1.662	.381	19.006	1	.000	5.271	2.497	11.128
Constant	-5.000	3.094	2.611	1	.106	.007		

a. Variable(s) entered on step 1: AGE, SEX, DIAGNOSIS, DM, HTN, smoking, BMI, WC, SBP, DBP, T.CHL, LDL, HDL, TGL, TCHDL, EAT3.4, CIMT.

Waist circumference (P < 0.001), LDL-C (P=0.039), EAT thickness (P <0.001) and CIMT (P <0.001) are independently predictive of CAD.

DISCUSSION

This study was conducted to assess the correlation between EAT thickness and the presence of obstructive CAD, severity of CAD and the correlation between SVD, DVD, TVD and left main involvement. It was also analysed for the ability to predict CAD and also the probability of using a combined CIMT and EAT thickness in predicting obstructive CAD. The association between EAT thickness and LV mass was assessed. EAT was assessed in correlation with the study population's baseline characteristics and risk factors.

EAT thickness in the study population

The mean EAT thickness was 4.04 ± 1.94mm. This is higher than the study of Ahn et al (9), but is lower when compared to many other studies [8, 10, 53]. The study population consisted of 28.7% with chronic stable angina, 26.2% with UA/NSTEMI and 45.1% with myocardial infarction. Interestingly the mean EAT thickness in infarction was slightly more than those with chronic stable angina and unstable angina (4.11mm Vs 4.07mm Vs 3.9mm respectively). However the difference did not reach statistical significance (P = 0.79). Unlike study by Jeong et al., which had a significant number of patients with unstable angina (51%), our study had a higher percent from infarction group [53].

18% had Single vessel disease and double and triple vessel disease was present in 25% in each group. The mean EATT was 4.73mm Vs 4.78mm Vs 5.23mm in the SVD Vs DVD Vs TVD respectively. Similar the previous reports EATT was not statistically different between this groups, P = 0.12 [9, 10].

EAT thickness compared to clinical characteristics and risk factors:

i. **Sex Vs EAT thickness**:

Of the study population of 244 patients, 190 were males and 54 were females, out of which 135 (71%) and 30(55.6%) respectively had significant CAD. This reflects an increased prevalence of CAD in males. Also it was noted that males had significantly higher EAT thickness compared to females (P = 0.034).

ii. **Age group Vs EAT thickness**:

Mean age of the study population was 53.05 ± 9.76 years. The mean age was not statistically different in males and females (P = 0.32). The patients were divided into three age groups of < 40 years, 40 – 60 years and > 60 years for analysis. The mean EAT thickness did not vary significantly between the age groups (P = 0.15).

iii. Diabetes, Hypertension and Smoking Vs EAT thickness

42.6% of the study group were diabetics. Diabetics had a mean EAT thickness of 4.51± 1.9mm compared 3.7± 1.9 which was statistically different, $P < 0.001$.

48.4% had hypertension and 38.9% were smokers. EAT thickness was statistically different among the within hypertensives and normotensives ($P = 0.64$). Similarly smokers and non smokers had an insignificant EAT thickness levels ($P = 0.584$).

iv. BMI Vs EATT

A total of 207 patients had normal BMI, with 32 patients (13%) overweight and 5 patients being obese (2%). All the obese patients had abnormal EAT levels. The EATT was significantly different between the groups, $P = 0.001$. However the correlation between the two parameters was weak, Pearson's R value = 0.291. This finding was similar to previous studies [8, 53, 56].

v. Waist circumference and EATT

When the cut off for waist circumference was set at 102cm for males and 88cm for females, there were 20 (8.2%) patients with increased waist circumference. However when the cut was set at a lower level 85cm for males and 80cm for females as studies have shown that

the cut for Indians should be set at a much lower level, there were 146 (59.4%) patients with increased waist circumference [96]. Both the cut off found a statistically significant correlation between EAT thickness and waist circumference. However the correlation was weak if cut off was set higher (r = 0.202) as compared to a better correlation when the cut off was lower (r = 0.559). These findings were similar to other studies [53, 56].

vi. LIPID PROFILE Vs EATT

- Total cholesterol was more than 200mg/dl in 78 participants (32%) with mean EAT of 4.38 ± 2.04. EATT was not statistically different between the groups, P=0.06.
- LDL was stratified into 4 groups with 32 patients (21.7%) having more than 160mg/dl. The mean EAT thickness was not statistically different between the sub groups, P = 0.41.
- HDL-C was low in 85 (34.8%) participants. EATT was 4.605 ± 1.91 in this group, which was statistically different (P=0.001).
- TGL was stratified into 3 groups with 65 (26.6%) having TGL more than 200 mg/dl. EATT was statistically different between the subgroups, P = 0.005

- TC/HDL ratio was also statistically different EATT between the subgroups, P = 0.007. 68 patients had a ratio less than 3.5 and 67 patients (27.5%) had more than 5.

The previous studies did not show any significant difference between any of the lipid profile parameters and EAT thickness [10, 53, 54]. In the present study the TGL, HDL and the TC/HDL ratio had statistically significant differences in EAT thickness. Total cholesterol and LDL cholesterol did have significant EATT variation.

vii. LV mass and Diastolic Dysfunction:

Previous studies have demonstrated that EAT thickness was associated strongly with left ventricular mass, left atrial size, and impaired left ventricular diastolic filling [50]. Similarly we also found a strong correlation between EATT, LV mass and diastolic dysfunction (P<0.001). However with linear regression we noted that only Diastolic dysfunction (P=0.004), along with waist circumference (P<0.001) and hypertension (P = 0.002) were independently predictive of EAT thickness.

EATT and CIMT: Correlation with presence and severity of CAD.

Various previous studies have found significantly varied cut off for EAT thickness from 3.0mm [9], 5.5mm [8], 6.3mm [12], 7.6mm [53] and 10mm [10]. Also in a study conducted on general Indian population it found

that the mean EATT was 2.6 ± 1.3 mm. This difference in EATT was possibly due to the different population, baseline characteristics and essentially different races. Hence in this present study we attempted to find out the cut off for EATT in predicting CAD in our population. As seen from the previous studies and also from the mean of Indian population, we set a lower limit cut off of 3mm for predicting CAD. The 75th percentile was 5.7mm and hence the arbitrary upper cut off was set at 6mm.

Similar to previous studies by Ahn et al and Jeong et al, we found a significant correlation between EAT thickness and CAD ($P < 0.001$, $r = 0.667$) [8, 53]. With an EATT cut off of 3mm it was noted to have a sensitivity of 90.9% and a specificity of 74.7% in predicting CAD. The negative predictive value was 79.7%. We also noted good correlation, $r = 0.657$. When the cut off was set at 4mm for predicting CAD, the specificity increased to 92.4%, but the sensitivity reduced to 71.5%. The correlation was average, $r = 0.575$. When the cut off was increased to 6mm, the sensitivity was poor at 25.5%, but the specificity was very good at 97.5%. The correlation however was weak, $r = 0.279$. The sensitivity and specificity was better than in previously noted by Toufan et al, with a cut off of 3mm [10].

As noted in previous studies, CIMT correlated to CAD, $P < 0.001$ [85]. The sensitivity (87.3 %) and specificity was (75.9 %) are almost

similar to EATT. Negative predictive value was better (91.7%). The correlation was also similar to EATT with a cut off of 3mm, r=0.704.

Receiver operating characteristics (ROC) curve was used to determine the high risk value Of EATT to predict CAD. EATT of 3.4mm was determined as a high risk value for predicting significant CAD with an85 % sensitivity and 78.5 % specificity.

We also wanted to find out if the combination of EATT and CIMT is a better predictor of significant CAD. We found that the sensitivity of the combined test was better than EATT or CIMT alone (92.1%), but the specificity was slightly less (73.4%). However, the negative predictive value was good (81.7%). The correlation between CAD and the combined test was good, r=0.675, P<0.001. This finding shows that the combined test could be a better predictor of significant CAD and also in patients with a negative test, it could effectively rule out CAD.

We also analyzed the ability of EATT to predict the severity of coronary atherosclerosis. We used the GENSINI score to correlate between the severity and EATT. The mean GENSINI score was 27.8 ± 28.9. When a cut off of 3.4mm was used the GENSINI score was significantly different between the two groups, 38.66 Vs 7.17, P < 0.001. In study by Burak et al, they found a good correlation between SYNTAX score and EAT thickness, r = 0.69, P <0.001[8]. Similarly in our study we found good correlation

between EATT and GENSINI score, r = 0.66, P<0.001. The combined test also showed a significant correlation with gensini score, r = 0.531, P<0.001. When multiple linear regression was used for testing GENSINI score it was found that only Diabetes (P = 0.43), Waist circumference (P = 0.017), BMI (P = 0.019), EATT (P < 0.001) and CIMT (P =0.003) were independently predictive of increased GENSINI score. Other parameters were not independently predictive of the severity of atherosclerosis.

Ahn et al, had shown that EAT of 3mm was independently predictive of CAD (OR 3.357, 95% CI 2.175 – 5.175, P <0.001) [9]. Similarly in the study by Jeong et al they found that showed that age (odds ratio (OR) 5.29, p=0.003), epicardial fat thickness (OR 10.53, p=0.004), diabetes (OR 8.06, p=0.006) and smoking (OR 14.65, p=0.015) were independent factors affecting significant coronary artery stenosis [53]. In our study we found that waist circumference (OR 7.63, P<0.001), LDL-C (OR 2.7, P=0.039), EATT >3.4mm (OR 5.71, P<0.001) and CIMT >1mm (OR 5.71, P<0.001) were independently predictive of CAD. Other risk factors or BMI were not independently predictive of CAD.

CONCLUSION

The average EAT thickness of the population is 4.04mm. The EAT thickness is significantly more in males. The EAT thickness varied significantly in diabetics compared to non diabetics. The EAT thickness was significantly higher in groups with higher BMI and increased waist circumference. The EAT was significantly higher in patients with increased triglycerides, increased TC/HDL ratio and reduced HDL levels. This confirms that EAT thickness can be reliably used as a marker of metabolic syndrome and quantify the severity of underlying metabolic disorder.

Increased EAT thickness was significantly more common in patients with obstructive CAD. Any value of EAT below 3mm was found to have very low specificity and with increasing cut off of up to 6mm we found that the specificity increased appreciably, but at the loss of sensitivity. An EAT thickness of 3.4mm was found to reliably predict the obstructive CAD in the study population with a balance between sensitivity and specificity.

We found that EAT thickness correlated significantly with the severity of atherosclerosis. An EAT thickness of 3.4mm was able to reliably predict patients with significantly coronary atherosclerosis.

However the mean EAT did not significantly between single, double or triple vessel disease and hence could not be used reliably to predict the same.

When compared to CIMT, EATT cut off of 3.4mm showed an almost similar sensitivity, specificity and negative predictive value. We analysed whether the combination of CIMT and EATT was better than either of the two in predicting CAD. We found out that the combined test had a better sensitivity and negative predictive value, with only a slightly lower specificity. Hence the combined test could be proposed as a reliable marker in predicting CAD.

Hence to conclude,

- The EATT assessed by transthoracic echo could be convincingly used as a screening test in predicting obstructive CAD.
- The EATT when compared to established atherosclerotic marker like CIMT was found to be equally effective as a screening tool.
- Use of EAT thickness measurement in routine practice could be of assistance in identifying patients at risk and guiding them in proper control risk factors and, if required, undergoing evaluations with invasive approaches.

- EATT cut value of 3.4mm was found to be the ideal cut off value in the Indian population for predicting CAD, with good accuracy.
- The combined test is a better predictor of CAD and hence could be used as clinical marker of CAD.

LIMITATIONS AND SCOPE FOR FUTURE STUDIES.

This was a single centre study; hence the results cannot be generalized to the Indian population. The analysis was also limited by the fact that the study enrolled only patients undergoing coronary angiogram. Transthoracic echo has certain limitations like poor acoustic window, which limits the usage in some patients. 2D echo will not be able to completely assess the whole of epicardial fat which is a 3 dimensional structure. A gold standard tool like MRI could used for comparison, due to cost constraints.

A large multi centric population based epidemiological study with follow up is necessary to ascertain the role of EAT thickness as an effective screening tool in patients suspected of having metabolic disorder and atherosclerotic disease.

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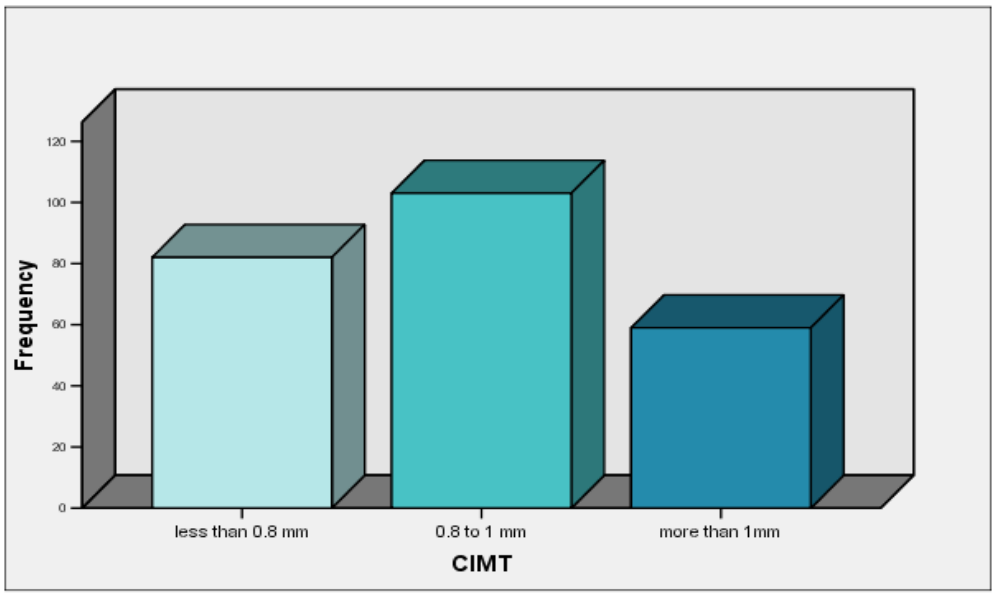
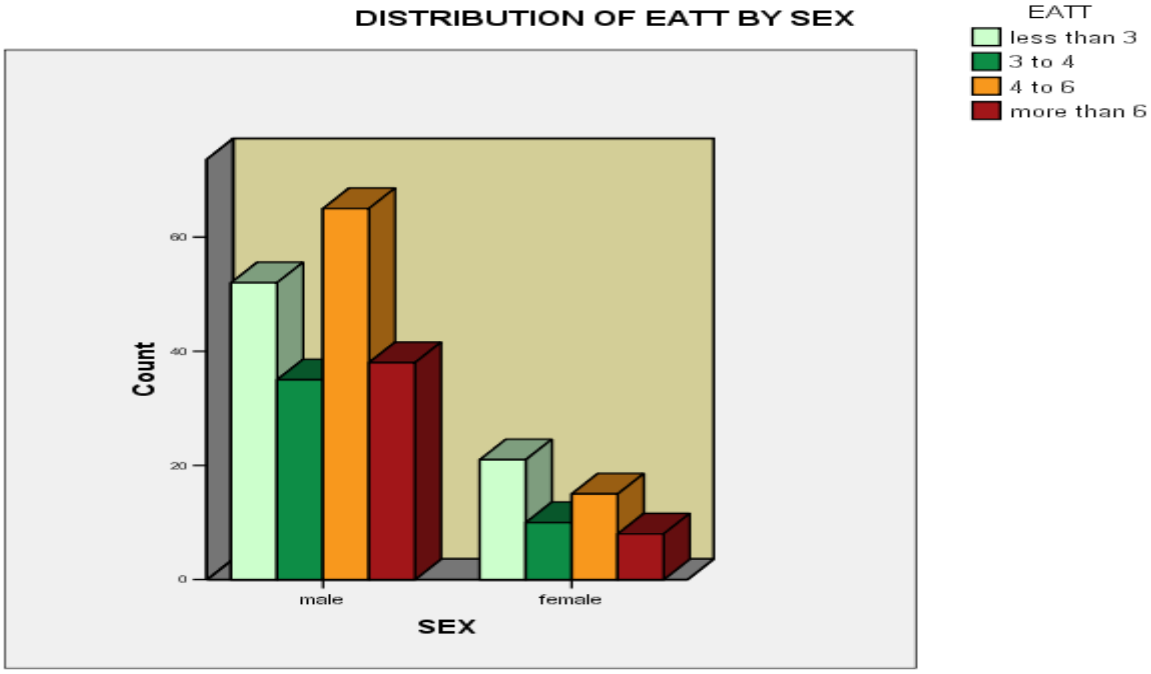
Distribution of EAT thickness

Distribution of CIMT



DISTRIBUTION OF EATT BY SEX



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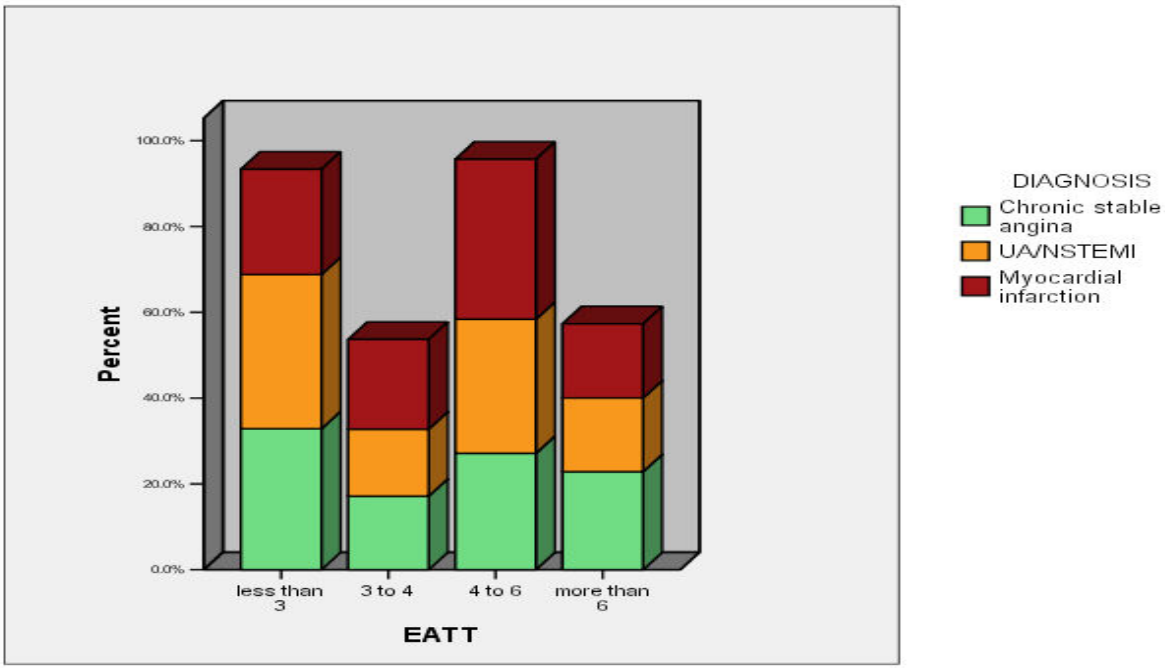
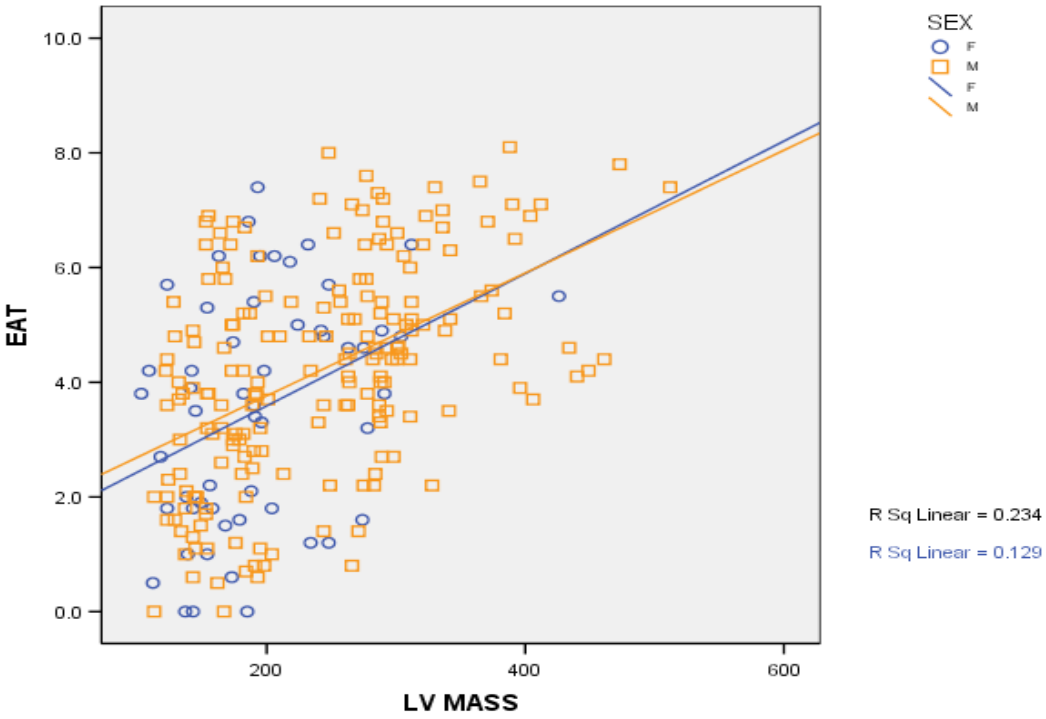
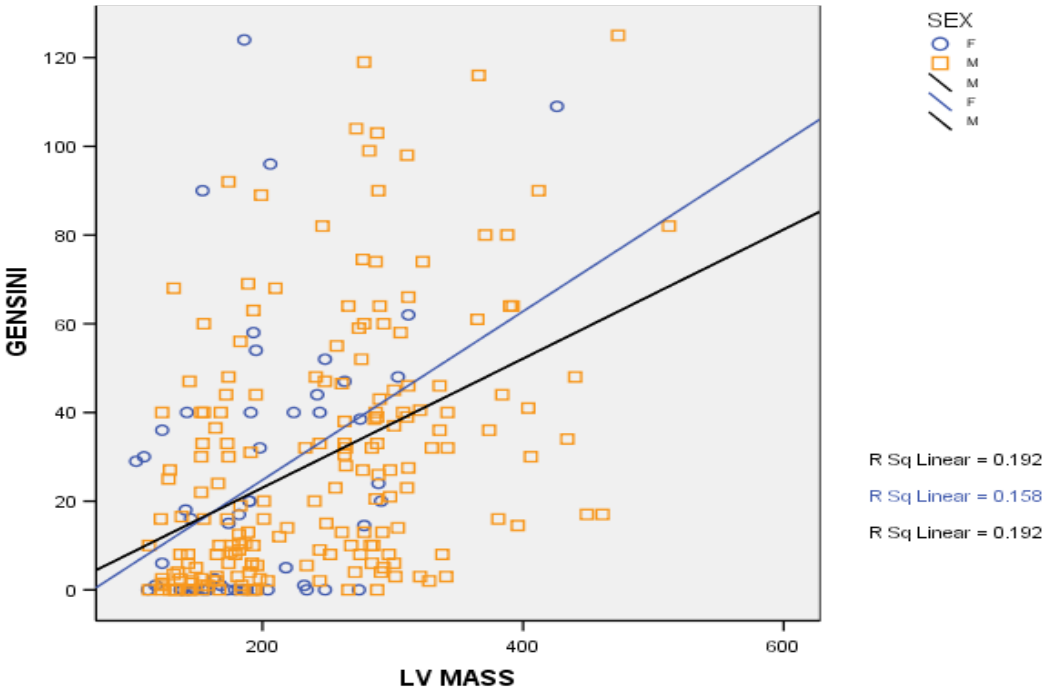
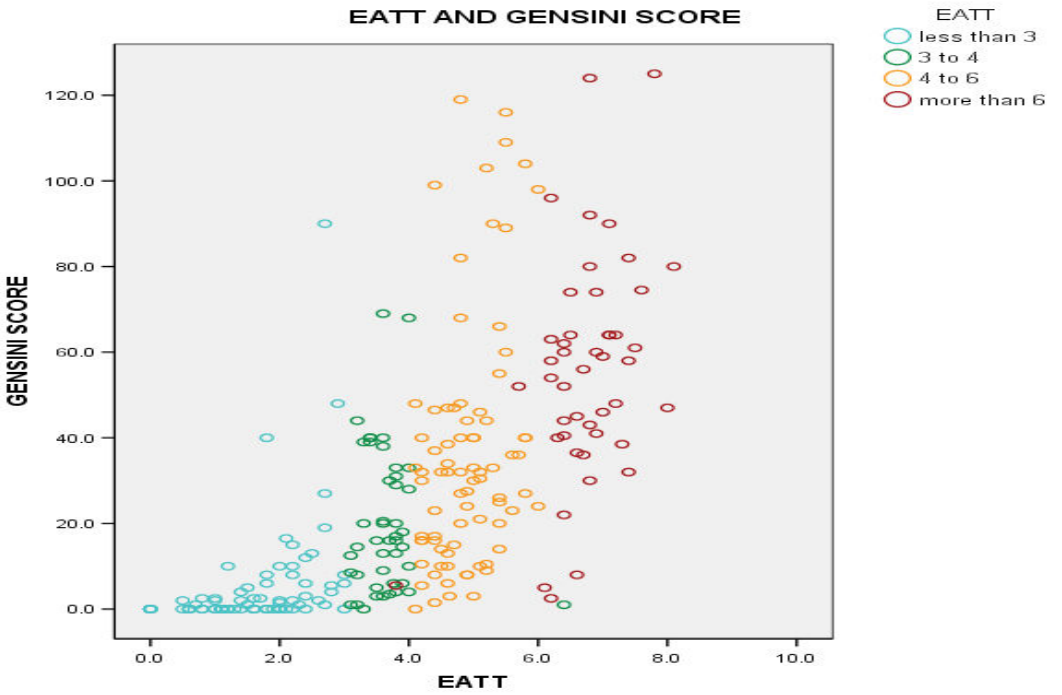


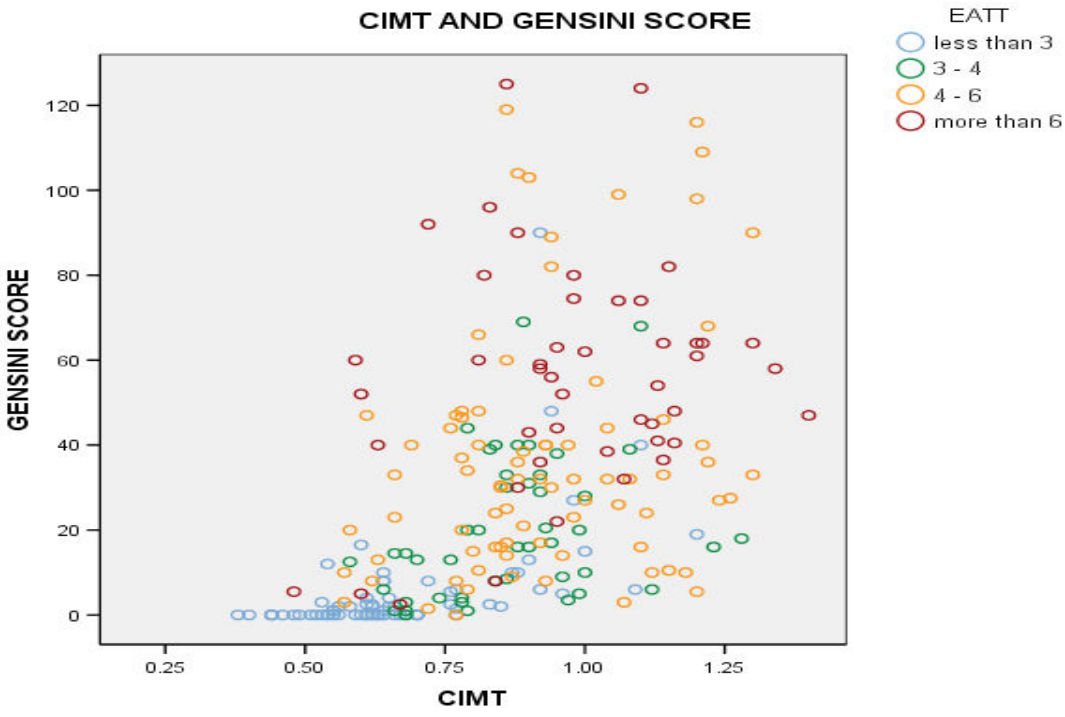
CORRELATION OF LV MASS AND EATT IN MALES AND FEMALES

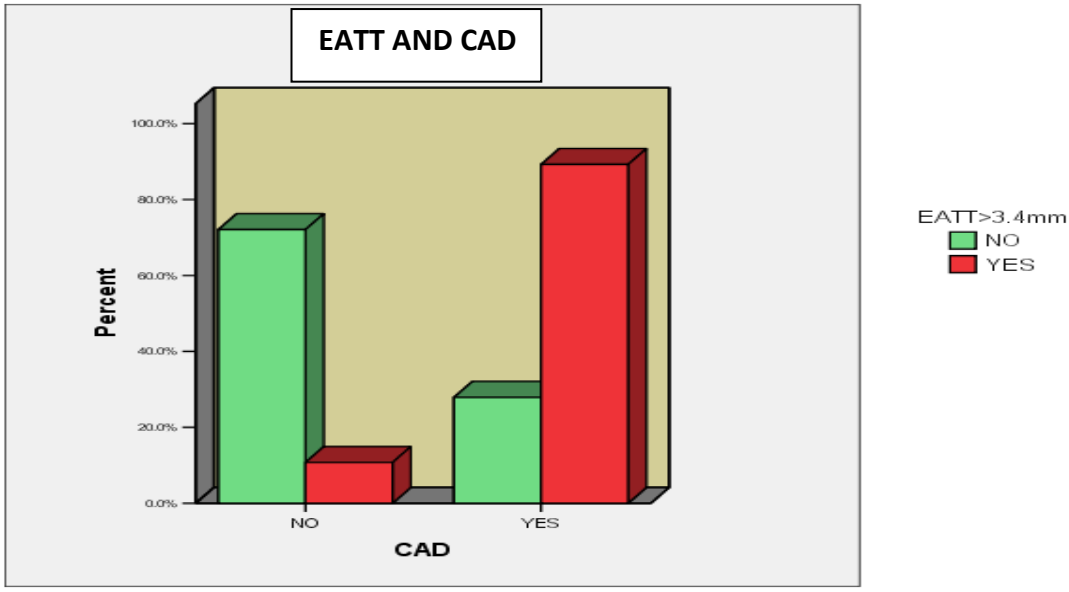


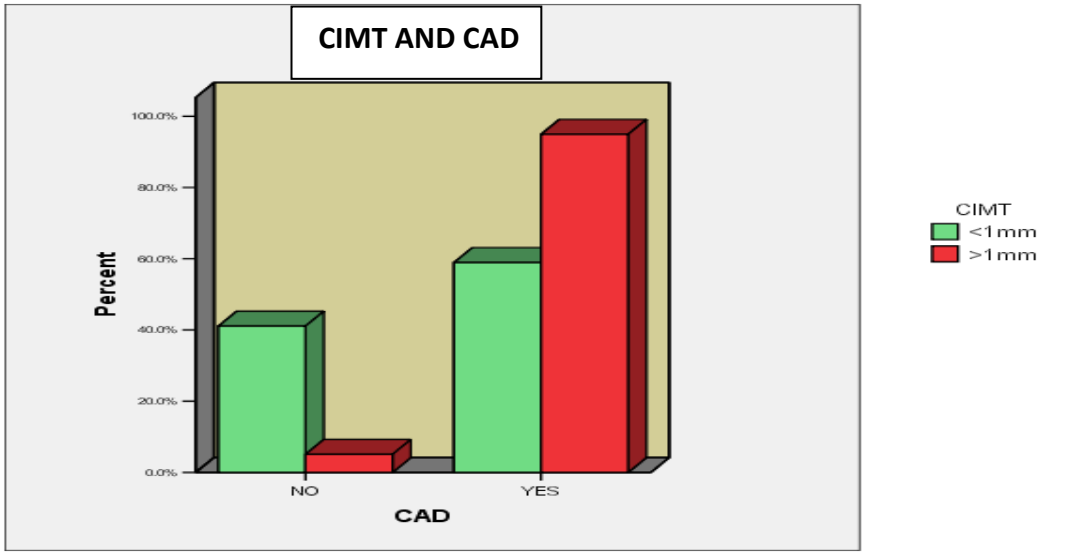
CORRELATION OF LV MASS AND GENSINI SCORE IN MALES AND FEMALES




EATT AND GENSINI SCORE
EATT
less than 3
3 to 4
4 to 6
more than 6
GENSINI SCORE
120.0
100.0
80.0
60.0
40.0
20.0
0.0
0.0
2.0
4.0
6.0
8.0
10.0
EATT


CIMT AND GENSINI SCORE
EATT
less than 3
3 - 4
4 - 6
more than 6
GENSINI SCORE
120
100
80
60
40
20
0
0.25
0.50
0.75
1.00
1.25
CIMT


EATT AND CAD
Percent
100.0%
80.0%
60.0%
40.0%
20.0%
0.0%
NO
YES
CAD
EATT>3.4mm
NO
YES


CIMT AND CAD
Percent
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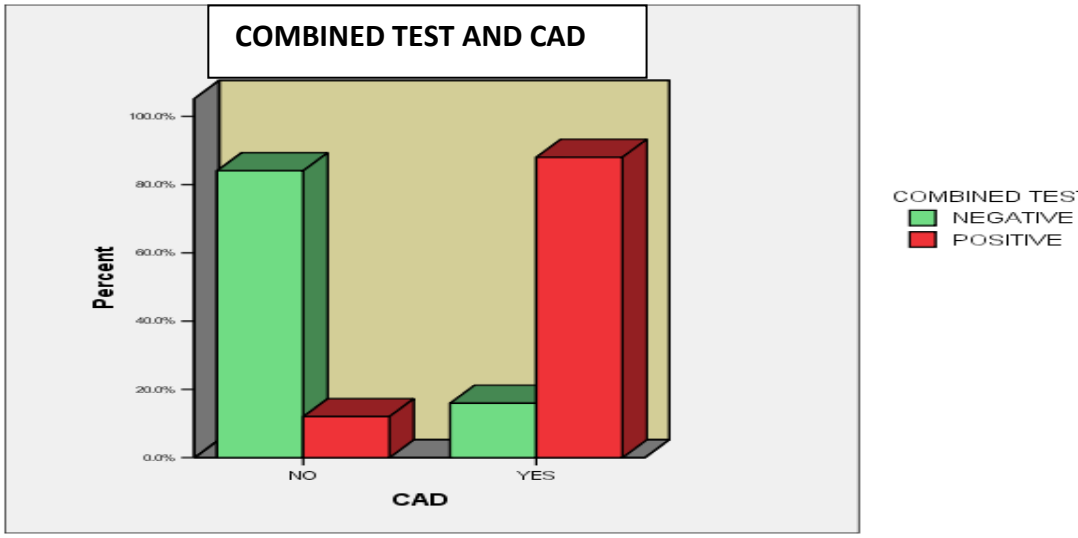
80.0%
60.0%
40.0%
20.0%
0.0%
NO
YES
CAD
CIMT
<1mm
>1mm


COMBINED TEST AND CAD
Percent
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80.0%
60.0%
40.0%
20.0%
0.0%
NO
YES
CAD
COMBINED TEST
NEGATIVE
POSITIVE

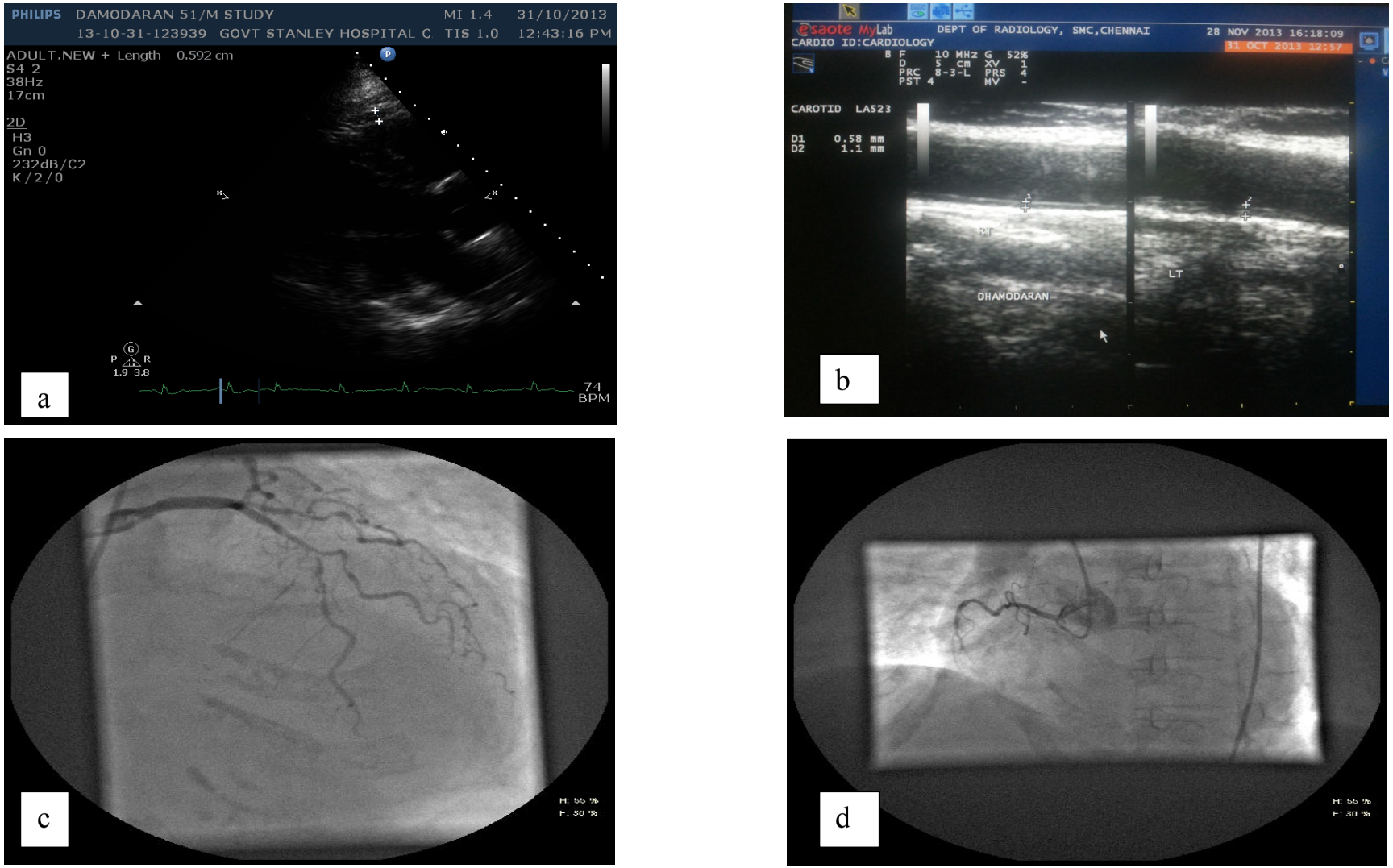

Figure showing increased EAT thickness (a) and CIMT (b) in patient with triple vessel disease (c & d).

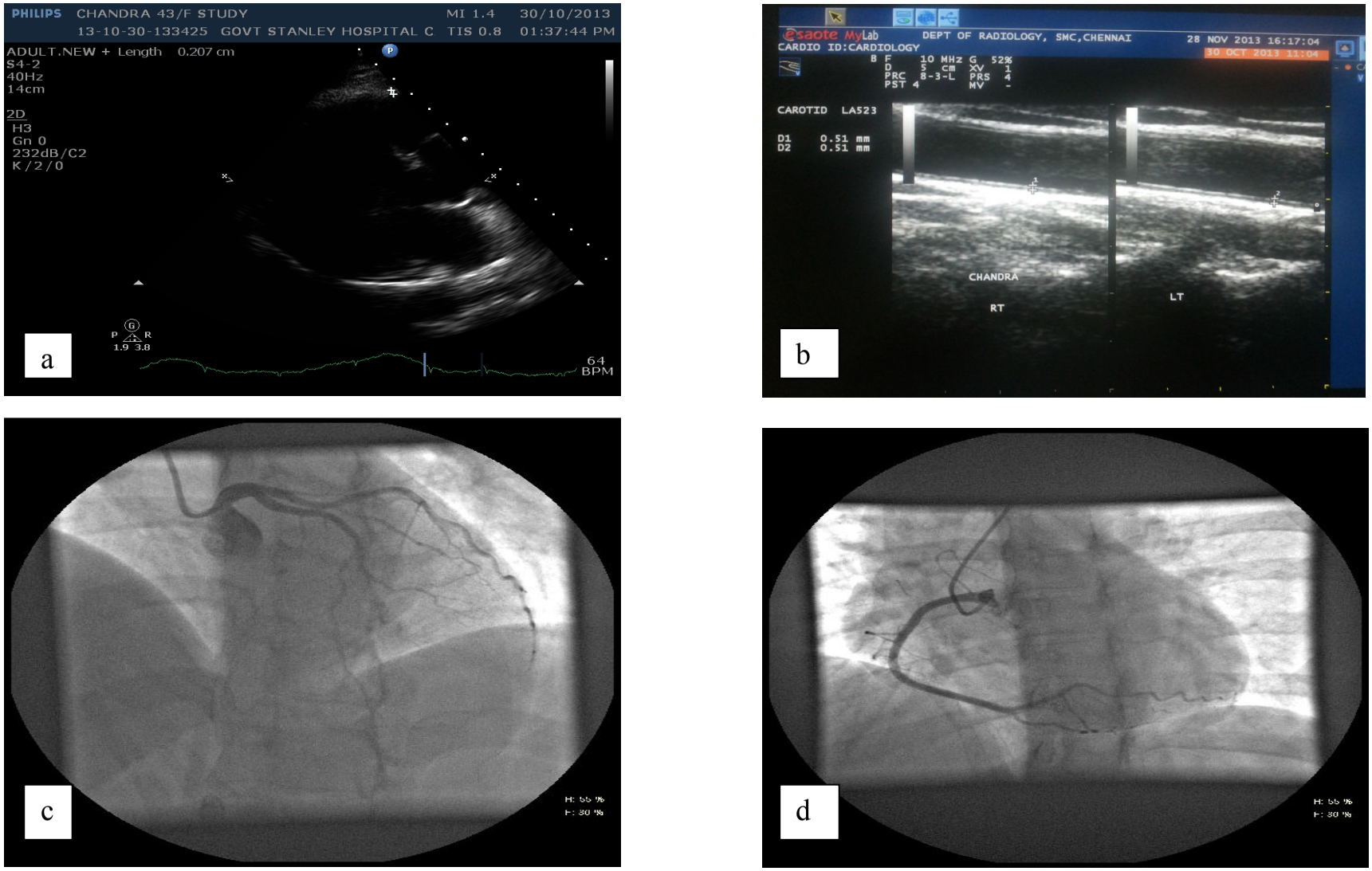

Figure showing normal EAT thickness (a) and CIMT (b) in patients with normal coronary angiogram (c & d).

INSTITUTIONAL ETHICAL COMMITTEE, STANLEY MEDICAL COLLEGE, CHENNAI-1

Title of the Work : A study of echocardiographic epicardial adipose tissue thickness and carotid intima medic thickness to predict severity of coronary artery disease

Principal Investigator : Dr.P.Vinodh Kumar

Designation : PG in D.M (Cardio)

Department : Department of Cardiology
Government Stanley Medical College,
Chennai-10

The request for an approval from the Institutional Ethical Committee (IEC) was considered on the IEC meeting held on 08.11.2013 at the Council Hall, Stanley Medical College, Chennai-1 at 2PM

The members of the Committee, the secretary and the Chairman are pleased to approve the proposed work mentioned above, submitted by the principal investigator.

The Principal investigator and their team are directed to adhere to the guidelines given below:

1. You should inform the IEC in case of changes in study procedure, site investigator investigation or guide or any other changes.
2. You should not deviate from the area of the work for which you applied for ethical clearance.
3. You should inform the IEC immediately, in case of any adverse events or serious adverse reaction.
4. You should abide to the rules and regulation of the institution(s).
5. You should complete the work within the specified period and if any extension of time is required, you should apply for permission again and do the work.
6. You should submit the summary of the work to the ethical committee on completion of the work.

20/1/14

MEMBER SECRETARY,
IEC, SMC, CHENNAI

A STUDY OF ECHOCARDIOGRAPHIC EPICARDIAL ADIPOSE TISSUE THICKNESS AND CAROTID INTIMA MEDIA THICKNESS TO PREDICT SEVERITY OF CORONARY ARTERY DISEASE

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INTRODUCTION

Coronary artery disease (CAD) is considered the leading cause of death in the world. In 2004, CAD was the cause of 7.2 million deaths (About 12% out of a total of about 60 million deaths) [1]. At the same time in India, CAD related deaths was the most common cause, leading to about 1.5 million deaths (14% out of a total of approx. 10 million deaths).

CAD now causes more death and disability in socioeconomically low- and middle-income countries, such as India. The CAD related death rate is increasing alarmingly when compared to high-income countries. CAD in low- and middle-income countries affects people at younger ages, compared to high-income countries [2]. Unadjusted CAD rates in India have ranged from about 1% to about 13%, in urban populations and about 1.6% to 7.4%, in rural populations [3]. In a study from urban population like Chennai the prevalence of CAD was 11% [4].

This alarming increase in CAD prevalence is a cause of concern. While the major aspect in dealing with CAD is identification and modification of risk factors, equally important is the ability to identify the individuals in early stage of CAD, before development of any adverse clinical event, chronic disability or death.

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PROFORMA

Name: **Age:** **Sex:**

Occupation: **DOA:** **IP NO:** **CD NO**

Address:

Diagnosis:

Risk Factors:

Male Gender: DM: Smoking: Dyslipidemia: HTN: F/H of CAD:

Examination:

Height: **Weight:** **BMI:** **Waist circumference:**

Vitals: - pulse: BP: SpO2:

CVS: RS:

ECG:

TMT (optional):

ECHO:

IVSd / IVSs: LVIDd: AO/LA diameter (M mode):

LV PWd/ PWs: LVIDs:

EF (TEICHOLZ):

RWMA:

Mitral valve: Aortic valve: Tricuspid valve:

PHT

Diastolic dysfunction: E/A: E/E':

RA/RV function: pericardial effusion:

LV Mass: 0.8 (1.04 ([LVIDD + PWTD + IVSTD] ^{3}- [LVIDD] ^{3})) + 0.6 g:

	Test 1	**Test 2**	**Test 3**	**Average**
EAT				

CAROTID INTIMA MEDIA THICKNESS:

BLOOD INVESTIGATIONS

Hb: RBS: UREA: CREATININE:

ELECTROLYTES - NA/K:

LIPID PROFILE:

Sr. CHOLESTEROL **Sr. TGL:** **Sr. LDLC:**

Sr. HDLC: **TC/HDL:**

CARDIAC CATHETERIZATION:

Coronary angiogram findings:

Gensini score:

Consent Form

I agree to participate in the study titled - "**A study of echocardiographic epicardial adipose tissue thickness and carotid intima media thickness to predict severity of coronary artery disease**"

I confirm that I have been told about this study in my mother tongue and have had the opportunity to ask question.

I understand that my participation is voluntary and I may refuse to participate at any time without giving any reason and without affecting my benefits.

I agree not to restrict the use of any data or results that arise from the study.

I agree to undergo the necessary investigation which is part of the study.

Name of the participant:

Signature / thumb impression:

Investigator:

நோயாளிகளுக்கான ஆலோசனை

மீளொளி முலம் இதய தசை சூழ் கொழுப்பு படிவங்களை அளந்து, இதயத்திற்கான தமனிகளின் கொழுப்பு படிவ அடைப்புகளை அளவீடு செய்வதுடன், மூளைக்கான தமனிகளின் உள்உறை சுவரின் தடிமனும் ஒப்பீடு செய்வதற்கான ஒரு செயல்முறை பரிசோதனை பற்றி நான் ஒரு ஆய்வு மேற்கொண்டு உள்ளேன்.

இந்த கண்காணிக்கப்பட்ட மருத்துவ ஆய்விற்கு தாங்களும் பதிவு செய்து தங்களது முழு ஒத்துழைப்பை நல்குமாறு தங்களை அன்புடன் கேட்டுகொள்கிறேன் .

நோயாளிகள் ஒப்புதல்

இந்த இருதய உட்புகுத்து பரிசோதனை மற்றும் மீளொலி பரிசோதனை பற்றி விளக்கப்பட்டது. அதனுடன் இதனால் ஏற்படகூடிய பக்க விளைவுகள் பற்றி மருத்துவரின் மூலம் தெரிந்துகொண்டேன்.

பரிசோதனை மற்றும் நடத்தப்படும் ஆய்வை பற்றி முழுமையாக மருத்துவர் விளக்கினார். நான் இந்த ஆய்வில் பங்கெடுக்க முழு மனதுடன் சம்மதம் தெரிவிகின்றேன் .

நோயாளியின் கையொப்பம்

ஒப்புதல் படிவம்

மீளொளி மூலம் இதய தசை சூழ் கொழுப்பு படிவங்களை அளந்து, இதயத்திற்கான தமனிகளின் கொழுப்பு படிவ அடைப்புகளை அளவீடு செய்வதுடன், மூளைக்கான தமனிகளின் உள்உறை சுவரின் தடிமனும் ஒப்பீடு செய்வதற்கான ஒரு செயல்முறை பரிசோதனை

நோயாளியின் ஒப்புதல் படிவம்

ஆராய்ச்சி நிலையம் : அரசு ஸ்டான்லி மருத்துவமனை, சென்னை 600001

பங்கு பெறுபவரின் பெயர் :

பங்கு பெறுபவரின் கையொப்பம் :

பங்கு பெறுபவர் இதனை ( ) குறிக்கவும்

மேலே குறிப்பிட்டுள்ள மருத்துவ ஆய்வின் விவரங்கள் எனக்கு விளக்கப்பட்டது. என்னுடைய சந்தேகங்களை கேட்கவும், அதற்கான தகுந்த விளக்கங்களை பெறவும் வாய்ப்பளிக்கப்பட்டது . ☐

நான் இந்த ஆய்வில் தன்னிச்சையாகத்தான் பங்குபெருகிரேன் .எந்த காரணத்தினாலோ எந்த சட்டசிக்கல்களுக்கும் உட்படாமல் நான் இந்த ஆய்வில் இருந்து விலகிக்கொள்ளலாம் என்று அறிந்து கொண்டேன். ☐

இந்த ஆய்வு சம்பந்தமாகவோ, இதை சார்ந்த மேலும் ஆய்வு மேற் கொள்ளும் போதும் இந்த ஆய்வில் பங்குபெறும் மருத்துவர் என்னுடைய மருத்துவ அறிக்கைகளை பார்ப்பதற்கு என் அனுமதி தேவையில்லை என அறிந்துகொள்கிறேன்.நான் ஆய்வில் இருந்து விலகிக்கிகொண்டாலும் இது பொருந்தும் என அறிந்தேன். ☐

இந்த ஆய்வின் மூலம் கிடைக்கும் தகவல்களையும் , பரிசோதனை முடிவுகளையும், மற்றும் சிகிச்சை தொடர்பான தகவல்களையும் மருத்துவர் மேற்கொள்ளும் ஆய்வில் பயன்படுத்திக்கொள்ளவும் அதை பிரசுரிக்கவும் என் முழு மனதுடன் சம்மதிக்கிறேன். ☐

இந்த ஆய்வில் பங்கு கொள்ள ஒப்புக்கொள்கிறேன். எனக்கு கொடுக்கப்பட அறிவுரைகளின் படி நடந்து கொள்வதுடன் இந்த ஆய்வை மேற்கொள்ளும் மருத்துவ அணிக்கு உண்மையுடன் இருப்பேன் என்றும் உறுதி அளிகின்றேன். என் உடல் நலம் பாதிக்கப்பட்டாலோ அல்லது எதிர்பாராத, வழக்திர்க்குமாறன நோய்க்குறி தென்பட்டாலோ உடனே அதை மருத்துவ அணிக்கு தெருவிப்பேன் என உறுதி அளிக்கிறேன். 

இந்த ஆய்வில் எனக்கு ரத்தம், சிறுநீர், எக்ஸ்ரே, ஸ்கேன், உட்பட அனைத்து பரிசோதனைகளையும் செய்து கொள்ள நான் முழு மனதுடன் சம்மதிக்கிறேன். 

பங்கேற்பவரின் கையொப்பம்..... .....இடம்.....தேதி .....

கட்டைவிரல் ரேகை.....

பங்கேற்பவரின் பெயர் மற்றும் விலாசம் .....

.....

ஆய்வாளரின் கையொப்பம்..... ..இடம்.....
தேதி.....

ஆய்வாளரின் பெயர் .....

S.No	NAME	AGE	SEX	DIAGNOSIS	DM	HTN	smoking	BMI	WC	SBP	DBP	EF	D D	LV MASS	EATT	CIMT	COMB	RBS	T. CHL	LDL-C	HDL-C	TGL	TC/HDL	Normal	Non-obst	SVD	DVD	TVD	100% cut off	Left main	GENSINI
1	Gunasundari	45	F	UA/ NSTEMI	N	Y	N	26.5	90	120	84	61	N	158	1.8	0.53	N	105	164	83.2	44	184	3.72	Y	N	N	N	N	N	N	0
2	Ayangaram	62	F	CSA	N	Y	N	24	86	130	90	72	2	186	6.8	1.1	Y	78	212	137.2	37	189	5.72	N	N	N	N	Y	Y	N	124
3	Thirupal	65	M	AWMI	Y	N	Y	23.5	81	126	82	43	1	288	5.2	0.9	Y	146	236	162.8	34	196	6.94	N	N	N	N	Y	Y	N	103
4	umapathy	52	M	UA/ NSTEMI	N	Y	Y	22.4	91	140	100	55	2	276	2.4	0.96	Y	112	241	161.2	47	164	5.12	N	N	Y	N	N	N	N	52
5	annamalai	60	M	IWMI	Y	N	N	25.4	103	132	78	46	N	174	5	0.94	Y	152	170	88.6	39	212	4.35	N	N	N	Y	N	N	N	30
6	Noorayesha	72	F	IWMI	Y	Y	N	21.6	91	120	70	39	2	193	7.4	1.34	Y	124	191	90	39	310	4.89	N	N	N	N	Y	Y	N	58
7	Sakthivel	68	M	AWMI	N	N	Y	24	77	134	80	37	1	210	4.8	1.22	Y	132	278	194.8	52	156	5.34	N	N	N	N	Y	Y	N	68
8	Muthumari	48	M	AWMI	Y	N	N	27.6	88	112	74	44	N	257	5.4	1.02	Y	178	211	128.4	54	143	3.90	N	N	Y	N	N	N	N	55
9	Nagaraj	48	M	UA/ NSTEMI	N	N	Y	28.1	93	126	80	48	N	166	6	1.11	Y	89	226	148	41	185	5.51	N	N	Y	N	N	N	N	24
10	Md. Kaliq sherrif	49	M	ASMI	N	Y	N	26.4	88	106	68	38	1	261	4.4	0.78	Y	92	188	101.2	56	154	3.35	N	N	N	N	Y	N	N	46.5
11	annappan	66	M	CSA	Y	Y	Y	22.8	80	130	70	60	1	196	2.8	0.76	N	190	259	183.2	42	169	6.16	N	Y	N	N	N	N	N	5.5
12	Kasi	55	M	UA/ NSTEMI	N	N	Y	23.5	79	120	70	61	N	204	1	0.66	N	64	168	85.8	52	151	3.23	N	Y	N	N	N	N	N	2
13	Elangovan	49	M	CSA	N	Y	N	23.5	98	110	60	75	1	244	1.4	0.56	N	94	177	97.4	51	143	3.47	N	Y	N	N	N	N	N	2
14	Mariyamma	65	F	IWMI	N	N	N	21.4	83	114	72	53	2	275	4.6	0.89	Y	87	168	72.8	48	236	3.5	N	N	Y	N	N	N	N	38.5
15	Veimurugan	47	M	ASMI	Y	N	Y	22.6	89	126	80	50	1	183	6.7	0.94	Y	81	276	184.2	41	254	6.73	N	N	N	N	Y	N	N	56
16	Nazeem Ahmeed	43	M	UA/ NSTEMI	N	Y	N	31.2	104	120	90	57	2	272	5.8	0.88	Y	120	312	212.4	32	338	9.75	N	N	N	N	Y	N	N	104
17	Selvaraj	55	M	IWMI	Y	N	N	21	77	112	80	48	1	182	4.2	0.81	Y	102	274	190.8	54	146	5.07	N	N	Y	N	N	N	N	10.5
18	Kanchana	61	F	CSA	Y	N	N	23.8	86	150	80	65	1	188	2.1	0.62	N	190	244	145.4	41	288	5.95	Y	N	N	N	N	N	N	0
19	Padmini	60	F	UA/ NSTEMI	N	Y	N	29.4	96	170	80	59	2	195	6.2	1.13	Y	118	175	82.8	37	276	4.72	N	N	N	Y	N	Y	N	54
20	Ranjan	57	M	IWMI	N	N	Y	22.1	81	138	78	44	N	174	3	0.77	N	83	188	102.4	52	168	3.61	N	Y	N	N	N	N	N	6
21	Udayakumar	43	M	CSA	Y	N	N	23.1	82	100	60	76	1	191	0.8	0.55	N	134	149	64.2	56	144	2.66	Y	N	N	N	N	N	N	0
22	Mohan raj	32	M	AWMI	N	N	Y	23.5	84	130	90	40	N	176	3.1	0.86	Y	115	178	82	38	290	4.68	N	Y	N	N	N	N	N	8.5
23	Malliga	54	F	CSA	N	Y	N	21.8	76	114	66	66	1	248	1.2	0.44	N	90	166	78.2	48	199	3.45	Y	N	N	N	N	N	N	0
24	A.V. Krishnan	65	M	UA/ NSTEMI	N	Y	N	22.1	75	160	88	56	1	328	2.2	0.62	N	102	230	141.2	42	234	5.47	N	Y	N	N	N	N	N	2
25	Anjalai	55	F	CSA	Y	Y	N	23.4	88	154	104	68	N	191	3.4	0.9	Y	164	176	91.8	55	146	3.2	N	N	N	N	Y	N	N	40
26	Govindaraj	51	M	IWMI	N	N	Y	23.5	78	130	84	54	1	187	5.2	1.15	Y	143	184	99	47	190	3.91	N	N	N	Y	N	N	N	10.5
27	Sivakumar	40	M	AWMI	N	N	Y	22.1	84	120	74	46	N	176	1.2	0.88	N	95	193	105	46	210	4.19	N	N	N	Y	N	N	N	10
28	Shankar	42	M	CSA	Y	N	N	24.3	87	140	90	71	N	284	2.4	0.92	N	178	231	133	36	310	6.41	N	Y	N	N	N	N	N	6
29	purushothaman	65	M	UA/ NSTEMI	Y	Y	N	23.3	86	154	88	66	N	271	1.4	0.65	N	147	178	91.6	42	222	4.23	N	Y	N	N	N	N	N	4
30	Raam bai	52	F	UA/ NSTEMI	N	Y	N	24.1	86	112	72	70	N	179	1.6	0.48	N	88	274	191.2	52	154	5.26	Y	N	N	N	N	N	N	0
31	Ganapathy	65	M	UA/ NSTEMI	Y	Y	N	23.8	92	130	84	59	2	288	4	0.92	Y	151	170	87.6	44	192	3.86	N	N	N	N	Y	N	Y	33
32	Adhilakshmi	52	F	UA/ NSTEMI	Y	N	N	23.7	84	132	80	61	1	224	5	0.93	Y	164	192	101	37	270	5.18	N	N	Y	N	N	N	Y	40
33	Sundaraj	55	M	CSA	Y	N	Y	22.3	92	120	78	64	2	248	8	1.4	Y	135	290	189	35	330	8.28	N	N	N	N	Y	N	N	47
34	Kenneth carron	65	M	AWMI	N	Y	Y	24.2	84	140	84	39	1	336	7	0.64	Y	101	274	187.2	57	149	4.80	N	N	N	N	Y	N	N	46
35	Durai pandi	50	M	UA/ NSTEMI	N	N	Y	21.6	78	110	80	52	1	193	4	1	Y	84	167	81.8	54	156	3.09	N	N	Y	N	N	N	N	10
36	Harikrishnan	65	M	CSA	Y	N	N	20.4	81	132	86	64	N	149	1.5	0.96	N	98	177	96.6	43	187	4.11	N	Y	N	N	N	N	N	5
37	Kumar	45	M	IWMI	N	N	Y	23.9	89	148	88	55	1	278	3.8	0.76	Y	112	288	204.4	34	248	8.47	N	N	Y	N	N	N	N	13
38	Govindasamy	59	M	UA/ NSTEMI	N	N	N	24.2	88	170	94	73	1	244	3.6	0.96	Y	76	184	85.6	40	292	4.6	N	N	N	Y	N	N	N	9
39	Govindharajan	44	M	PWMI	N	N	Y	23.5	82	154	90	58	N	123	1.6	0.83	N	85	178	95.6	56	132	3.17	N	Y	N	N	N	N	N	2.5
40	Kesavan	55	M	IWMI	N	Y	N	23.1	83	100	70	56	N	146	2	0.58	N	69	140	57.4	55	138	2.54	N	Y	N	N	N	N	N	2
41	Ramaiyan	59	M	UA/NSTEMI	N	Y	N	22.6	83	150	90	63	1	129	4.8	1	Y	87	260	177	50	165	5.2	N	N	N	N	Y	N	N	27
42	Baskar	62	M	AWMI	Y	Y	N	21.8	85	136	84	62	2	241	7.2	1.16	Y	290	292	210	43	195	6.79	N	N	Y	N	N	Y	N	48
43	Sivakumar	33	M	UA/NSTEMI	N	N	Y	22.8	79	120	70	50	N	113	2	0.77	Y	105	189	110.2	52	134	3.63	N	N	Y	N	N	N	N	10
44	Thilagavathy	38	F	AWMI	N	Y	N	25.8	86	140	80	48	1	182	3.8	0.94	Y	135	190	110.6	51	142	3.72	N	N	N	Y	N	N	N	17
45	Elango	60	M	AWMI	Y	N	N	27.4	94	130	84	46	1	301	4.4	0.78	Y	310	241	164.4	41	178	5.87	N	N	N	Y	N	N	Y	37
46	Selvi	45	F	UA/NSTEMI	N	Y	N	23.6	79	120	68	60	N	173	0.6	0.61	N	90	138	58.6	51	142	2.70	Y	N	N	N	N	N	N	0
47	Chitti babu	54	M	CSA	N	N	Y	23	90	122	70	70	1	193	6.2	0.95	Y	102	290	206.8	42	206	6.90	N	N	N	N	Y	N	N	63

48	Sathyavathi	63	F	CSA	Y	N	N	26.4	91	128	70	67	N	185	0	0.55	N	114	252	168.2	47	184	5.36	Y	N	N	N	N	N	N	0
49	Gurunathan	53	M	IWMI	N	N	Y	24.2	83	134	80	47	N	158	3.1	0.79	Y	78	151	70	42	195	3.59	N	Y	N	N	N	N	N	1
50	Rajan	47	M	UA/NSTEMI	N	N	Y	22.1	80	110	74	58	1	213	2.4	0.54	N	89	244	163.8	50	151	4.88	N	N	Y	N	N	N	N	12
51	Kandasamy	57	M	ASMI	N	Y	N	23.4	77	130	90	40	1	263	4.1	0.66	N	102	232	147.2	58	134	4.0	N	N	N	N	Y	N	N	33
52	Dukambar	48	M	AWMI	N	Y	Y	20.8	80	130	76	39	N	173	4.2	1.1	Y	92	172	89	55	140	3.12	N	N	N	Y	N	N	N	16
53	Gopalakrishnan	27	M	AWMI	N	N	Y	21.5	80	116	74	40	N	198	0.8	0.62	N	95	154	72.4	54	138	2.85	N	Y	N	N	N	N	N	2.5
54	Malliga	57	F	AWMI	N	Y	N	22.4	89	140	94	37	2	289	4.9	0.84	Y	120	181	96.2	38	234	4.76	N	N	N	Y	N	N	N	24
55	Sundaramoorthy	65	M	CSA	Y	Y	N	24	90	154	88	66	3	191	3.8	0.9	Y	152	236	158.2	43	174	5.48	N	N	N	N	Y	N	N	31
56	Prabu	27	M	UA/NSTEMI	N	N	Y	23.6	74	98	70	58	N	143	1.3	0.4	N	131	176	97.6	44	172	4.0	Y	N	N	N	N	N	N	0
57	Koil pillai	50	M	UA/NSTEMI	Y	N	N	28.4	96	166	72	68	1	278	5.5	0.86	Y	174	290	208	40	210	7.25	N	N	N	N	Y	N	Y	60
58	Ramachandran	65	M	CSA	N	Y	Y	24.1	86	130	82	75	2	461	4.4	0.92	Y	85	276	194	41	205	6.73	N	N	Y	N	N	N	N	17
59	Vedhavalli	60	M	CSA	N	Y	N	24.5	91	160	90	72	1	311	3.4	1.08	Y	81	268	183	56	145	4.78	N	N	N	Y	N	N	Y	39
60	Ravichandran	47	M	UA/NSTEMI	Y	N	N	27.5	93	130	82	61	1	165	3.2	0.84	Y	146	192	113.6	45	167	4.26	N	N	N	Y	N	N	N	8
61	Annamalai	54	M	AWMI	N	N	Y	23	85	110	74	46	N	153	1.8	1.1	N	78	256	177.2	42	184	6.09	N	N	N	N	Y	N	N	40
62	Anand babu	30	M	AWMI	N	N	Y	24.2	94	172	90	44	2	388	8.1	0.82	Y	91	270	191.2	40	194	6.75	N	N	Y	N	N	Y	N	80
63	Muniyammal	70	F	CSA	Y	N	N	22.7	86	130	82	67	N	190	5.4	0.58	N	177	271	188	57	130	4.75	N	N	N	Y	N	N	N	20
64	Liston femandez	50	M	IWMI	N	Y	Y	23.8	81	160	90	42	1	263	3.6	0.95	Y	98	242	159.8	53	146	4.56	N	N	N	N	Y	N	Y	38
65	puviarasu	64	M	UA/NSTEMI	N	N	Y	24.5	97	116	72	64	N	277	5.8	1.24	Y	76	191	109.8	42	196	4.54	N	N	N	Y	N	N	N	27
66	Mani	58	M	IWMI	N	Y	N	21.8	79	120	82	46	1	249	2.2	0.52	Y	69	270	181.4	40	243	6.75	N	N	N	N	Y	N	N	15
67	Bommi	65	F	AWMI	Y	N	N	22.9	84	124	70	39	N	123	1.8	1.09	N	124	206	125.8	39	206	5.28	N	Y	N	N	N	N	N	6
68	Shajahan	62	M	CSA	N	Y	N	29	100	152	88	70	2	311	6	1.2	Y	88	288	202	33	265	8.72	N	N	N	N	Y	N	N	98
69	Sundarajan	43	M	UA/NSTEMI	N	N	Y	21.4	85	144	84	66	N	144	2	0.56	N	90	265	186.2	52	134	5.09	Y	N	N	N	N	N	N	0
70	Loganathan	42	M	IWMI	N	Y	Y	20.7	81	130	70	49	1	134	1.4	0.64	N	72	192	114.2	49	144	3.91	Y	N	N	N	N	N	N	0
71	Shazadi begum	56	F	UA/NSTEMI	Y	Y	N	22.8	80	126	68	71	1	156	2.2	0.7	N	116	185	108.2	46	154	4.021	Y	N	N	N	N	N	N	0
72	Varalakshmi	44	F	CSA	Y	N	N	21.5	83	132	78	68	N	143	1.8	0.59	N	107	282	201.6	55	127	5.12	Y	N	N	N	N	N	N	0
73	Sagayairudaiya raj	46	M	AWMI	N	Y	N	24.1	89	112	80	40	2	293	3.5	0.99	Y	101	232	149	54	145	4.29	N	Y	N	N	N	N	N	5
74	Raju	54	M	CSA	Y	Y	N	27	98	100	70	65	2	266	7.1	1.14	Y	86	168	84.2	45	194	3.73	N	N	N	N	Y	Y	N	64
75	Hariharakrishnan	65	M	UA/NSTEMI	N	N	Y	23	77	90	60	64	1	301	4.6	0.79	Y	99	258	173	42	215	6.14	N	Y	N	N	N	N	N	6
76	periyasamy	47	M	AWMI	N	N	Y	22.5	82	100	80	42	N	137	1	0.61	N	69	178	94.6	56	137	3.17	N	Y	N	N	N	N	N	2.5
77	Subulakshmi	50	F	UA/NSTEMI	N	Y	N	21	85	100	74	62	N	149	1.9	0.67	N	91	194	111.8	47	176	4.12	Y	N	N	N	N	N	N	0
78	Chellaiyan	70	M	CSA	N	N	Y	21.8	78	110	78	66	2	292	4.6	0.63	N	108	248	166.8	53	141	4.67	N	N	N	Y	N	N	N	13
79	Munusamy	56	M	CSA	Y	N	N	32	105	166	86	68	2	293	6.4	0.81	Y	148	212	135	39	190	5.43	N	N	N	N	Y	N	N	60
80	Annamalai	55	M	AWMI	N	N	Y	23.5	89	160	90	38	1	263	5.1	0.85	Y	90	255	179	44	160	5.79	N	N	N	N	Y	N	N	30.5
81	Thangavelu	70	M	CSA	Y	Y	N	21.3	85	154	88	66	2	301	6.6	1.12	Y	162	265	182.4	55	138	4.81	N	N	N	Y	N	N	N	45
82	Vijaya	40	F	AWMI	Y	Y	N	22	77	120	70	42	N	204	1.8	0.38	N	137	166	81.6	58	132	2.86	Y	N	N	N	N	N	N	0
83	Vincent	40	M	IWMI	N	Y	Y	23.8	90	100	70	48	1	164	6.6	1.14	Y	97	229	148.4	52	143	4.40	N	N	Y	N	N	Y	N	36.5
84	Ramachandran	65	M	CSA	N	Y	Y	23.2	88	146	90	71	2	412	7.1	0.88	Y	128	240	161.8	41	186	5.85	N	N	N	Y	N	Y	N	90
85	Govindaraj	44	M	AWMI	N	N	N	21.8	86	140	84	34	1	308	5	0.93	Y	132	323	238.2	54	154	5.98	N	N	N	N	Y	N	N	40
86	Durai	54	M	AWMI	Y	Y	N	20.6	81	126	70	38	1	183	2.7	1.2	N	294	264	180.4	57	133	4.63	N	N	N	N	Y	N	N	19
87	Pearson	53	M	CSA	N	Y	N	24.8	96	110	80	62	2	153	6.4	0.95	Y	97	252	167.6	40	222	6.30	N	N	N	N	Y	N	N	22
88	Bowsiya	50	F	AWMI	N	N	N	22.3	84	120	84	46	N	174	4.7	0.8	Y	78	155	75.8	48	156	3.22	N	N	N	Y	N	N	N	15
89	Ahmed jamal	53	M	UA/NSTEMI	Y	N	N	23.8	91	138	74	66	1	290	7.2	1.2	Y	171	148	65.6	56	132	2.64	N	N	N	N	Y	N	N	64
90	Rajaram	45	M	UA/NSTEMI	N	N	Y	21	79	110	86	56	N	133	2.4	0.6	N	75	281	203.4	42	178	6.69	Y	N	N	N	N	N	N	0
91	Balakrishnan	55	M	IWMI	N	N	N	23	87	154	88	46	2	384	5.2	1.04	Y	69	232	153.4	36	213	6.44	N	N	N	Y	N	N	N	44
92	Govinthan	61	M	UA/NSTEMI	Y	N	N	22.8	88	166	74	70	2	330	7.4	1.07	Y	92	276	170.6	35	352	7.88	N	N	Y	N	N	N	Y	32
93	Amirtham	65	F	CSA	N	Y	N	26	94	150	84	65	2	312	6.4	1	Y	124	230	152.2	41	184	5.60	N	N	N	N	Y	N	N	62
94	Naatiyaoli	50	F	UA/NSTEMI	Y	Y	N	21	76	124	80	66	1	154	5.3	1.3	Y	181	188	106	49	165	3.83	N	N	N	N	Y	Y	N	90
95	Annamalai	60	M	CSA	Y	Y	Y	21.6	79	130	72	62	1	173	5	1.14	Y	201	184	101.8	55	136	3.34	N	N	N	Y	N	N	N	33

96	Balaji raman	30	M	IWMI	N	N	Y	22.4	80	120	76	45	N	184	0.7	0.55	N	102	165	84.2	45	179	3.66	N	Y	N	N	N	N	N	1
97	Durai	55	M	CSA	Y	N	Y	24	91	144	78	58	1	278	4.8	0.86	Y	146	291	199.4	39	263	7.46	N	N	N	Y	N	N	N	119
98	Chandra	60	F	IWMI	N	N	N	23	86	120	76	43	2	263	4.6	0.61	N	93	270	193	43	170	6.27	N	N	N	N	Y	N	N	47
99	Senkolmary	48	F	CSA	Y	Y	N	22.1	80	112	70	68	1	274	1.6	0.68	N	142	273	192.2	52	144	5.25	Y	N	N	N	N	N	N	0
100	Murugan	43	M	UA/NSTEMI	Y	N	Y	35.5	110	110	80	58	1	174	6.8	0.72	Y	176	176	84	39	265	4.51	N	N	N	N	Y	N	N	92
101	kodalingam	52	M	IWMI	Y	Y	Y	23.4	89	130	70	44	2	298	5.1	0.89	Y	192	154	75.2	44	174	3.5	N	N	N	N	Y	N	N	21
102	Saravanan	35	M	AWMI	N	N	Y	22.8	84	104	72	39	N	201	4.8	0.78	Y	98	222	141.4	56	123	3.96	N	N	Y	N	N	N	N	20
103	Ajees sahib	35	M	IWMI	N	N	Y	26.1	96	130	90	46	N	449	4.2	0.86	Y	101	238	160	36	210	6.61	N	N	Y	N	N	N	N	17
104	Samuel jayachandran	41	M	UA/NSTEMI	Y	N	Y	24.6	98	142	90	70	2	311	4.4	0.66	N	116	274	185.4	38	253	7.21	N	N	N	Y	N	N	N	23
105	Suseela	64	F	CSA	N	Y	N	22.7	78	130	82	66	1	137	0	0.44	N	78	175	97.8	46	156	3.80	Y	N	N	N	N	N	N	0
106	Sagira bee	57	F	CSA	N	Y	N	23.9	76	100	70	68	N	145	3.5	1.23	Y	76	262	182.2	47	164	5.57	N	N	N	Y	N	N	N	16
107	Vasantha	57	F	CSA	Y	Y	N	23.1	87	120	80	62	1	198	4.2	0.98	Y	170	266	183.6	48	172	5.54	N	N	N	N	Y	N	N	32
108	Manovathy	50	F	UA/NSTEMI	N	N	N	27.6	90	130	90	59	1	242	4.9	0.76	Y	95	192	110.8	42	196	4.57	N	N	N	N	Y	N	N	44
109	Krishnan	55	M	IWMI	Y	N	Y	24.6	94	110	70	48	1	155	6.9	0.59	N	188	243	163.8	36	216	6.75	N	N	N	N	Y	N	N	60
110	Subaramiah	70	M	CSA	Y	Y	N	23.1	87	174	88	67	2	287	3.4	0.84	Y	167	196	117	38	205	5.15	N	N	N	N	Y	N	Y	40
111	Elangovan	50	M	IWMI	N	N	Y	24.2	95	110	70	43	1	234	4.2	1.2	Y	118	234	160.8	42	156	5.57	N	Y	N	N	N	N	N	5.5
112	Antony	46	M	IWMI	N	Y	N	23	89	132	80	48	2	256	5.6	0.98	Y	80	215	141.4	34	198	6.32	N	N	Y	N	N	N	N	23
113	Palanivel	67	M	IWMI	Y	N	N	22.5	84	124	74	50	N	138	2.1	0.6	N	241	181	100.2	54	134	3.35	N	N	N	Y	N	N	N	16.5
114	Guna	40	F	AWMI	Y	N	N	21.2	74	130	70	46	1	141	3.9	1.28	Y	190	244	163.8	50	151	4.88	N	N	N	Y	N	N	N	18
115	Magimaidoss	54	M	CSA	Y	Y	N	22.8	87	120	70	62	1	374	5.6	0.88	Y	187	176	97.8	43	176	4.09	N	N	N	Y	N	N	Y	36
116	Ravi	50	M	AWMI	N	N	Y	21.6	86	114	76	44	N	128	5.4	0.86	Y	93	183	103	52	140	3.51	N	N	N	Y	N	N	N	25
117	Kotti	42	M	AWMI	N	N	Y	20.8	79	110	70	39	N	162	0.5	0.85	N	103	174	90.8	54	146	3.22	N	Y	N	N	N	N	N	2
118	Paulraj	61	M	UA/NSTEMI	N	N	Y	25.6	90	130	74	67	2	274	7	0.92	Y	117	210	131.2	41	189	5.12	N	N	N	N	Y	Y	N	59
119	Beer Mohameed	52	M	AWMI	Y	N	N	23.4	85	160	90	50	1	268	5.1	1.12	Y	211	265	184.4	52	143	5.09	N	N	Y	N	N	N	N	10
120	Kasturi	60	F	AWMI	Y	N	N	24.1	88	170	100	44	2	206	6.2	0.83	Y	241	187	105.6	38	217	4.92	N	N	N	N	Y	N	Y	96
121	Murugan	40	M	AWMI	N	Y	Y	22.5	88	130	90	40	N	123	4.4	0.72	Y	92	273	194	41	190	6.65	N	Y	N	N	N	N	N	1.5
122	Joseph	52	M	UA/NSTEMI	Y	Y	N	21.8	81	132	70	49	1	132	3.7	0.97	Y	180	158	77.2	50	154	3.16	N	Y	N	N	N	N	N	3.5
123	Vasantha	40	F	CSA	Y	N	N	22.1	80	142	82	67	N	234	1.2	0.61	N	151	179	101.2	51	134	3.50	Y	N	N	N	N	N	N	0
124	Gyanaprakasam	63	M	AWMI	Y	N	N	29.7	101	128	70	45	1	312	4.9	1.26	Y	256	310	219.8	37	266	8.37	N	N	N	Y	N	N	N	27.5
125	Kumaresan	57	M	CSA	Y	Y	N	24.8	94	150	94	48	2	473	7.8	0.86	Y	119	272	182.4	34	278	8.0	N	N	N	N	Y	Y	Y	125
126	Chanrdrasekar	40	M	UA/NSTEMI	N	N	Y	21	78	116	78	60	N	122	4.2	0.85	Y	82	182	98.6	55	142	3.30	N	N	N	Y	N	N	N	16
127	Visalakshi	45	F	AWMI	N	Y	N	20.6	78	104	70	40	1	143	0	0.63	N	76	169	87.4	56	128	3.017	Y	N	N	N	N	N	N	0
128	Kanthavelu	45	M	IWMI	Y	N	Y	27.5	98	114	68	47	2	290	6.8	0.9	Y	198	275	178.4	37	298	7.43	N	N	N	Y	N	N	N	43
129	Murugesan	61	M	UA/NSTEMI	Y	N	N	22.3	84	122	70	64	1	182	5.2	0.87	Y	137	161	69.8	38	266	4.23	N	N	Y	N	N	N	Y	9
130	Ramesh	47	M	IWMI	N	Y	Y	22.8	82	178	100	44	1	381	4.4	0.84	Y	98	260	174.8	59	131	4.40	N	N	N	Y	N	N	N	16
131	Dinakar	66	M	CSA	Y	Y	N	24	94	164	106	65	2	284	4.6	1.04	Y	179	171	90.6	43	187	3.97	N	N	Y	N	N	N	N	32
132	satyakumar	45	M	IWMI	N	N	Y	21	76	132	74	46	N	143	3.9	1.12	Y	101	184	106	52	130	3.53	N	Y	N	N	N	N	N	6
133	Mani	62	M	IWMI	Y	Y	N	22.8	81	174	90	44	1	298	2.7	0.72	Y	202	193	116.6	40	182	4.82	N	N	N	Y	N	N	N	27
134	Muthuvel	42	M	IWMI	N	Y	Y	31.7	102	132	76	40	2	302	4.63	0.57	N	92	283	189.8	38	276	7.44	N	Y	N	N	N	N	N	3
135	Goush Basha	49	M	IWMI	N	N	Y	23.8	84	114	84	42	N	143	0.6	0.7	N	84	249	169.6	41	192	6.07	Y	N	N	N	N	N	N	0
136	Udayakumar	55	M	AWMI	N	Y	N	23.3	86	140	70	39	1	192	3.76	0.64	N	90	172	94	46	160	3.73	N	N	Y	N	N	N	N	6
137	Krishnan	62	M	CSA	N	N	N	22.1	74	100	80	67	N	181	2.4	0.53	N	76	178	98.4	56	118	3.17	N	Y	N	N	N	N	N	3
138	Selvam	57	M	CSA	Y	N	Y	24	88	120	70	65	N	323	6.9	1.06	Y	123	210	129.8	41	196	5.12	N	N	N	N	Y	N	N	74
139	Shankar	59	M	UA/NSTEMI	N	Y	N	21.7	79	150	90	58	1	288	4.1	0.77	Y	82	158	74.6	40	217	3.95	N	Y	N	N	N	N	N	0
140	Balasubramaniyam	45	M	UA/NSTEMI	N	N	Y	22.6	81	110	80	60	N	195	3.2	0.79	Y	109	236	159.4	52	123	4.53	N	N	Y	N	N	N	N	44
141	Paneer selvam	60	M	UA/NSTEMI	Y	N	Y	24.1	89	120	74	61	1	289	2.7	0.92	N	212	240	151.6	38	252	6.31	N	N	N	N	Y	N	N	90
142	Zahirunisha	40	F	CSA	Y	N	N	21.8	76	130	84	64	N	244	4.8	0.69	N	234	180	100.2	52	139	3.46	N	N	N	N	Y	N	Y	40
143	Susairaj	60	M	CSA	N	Y	N	23.1	83	144	70	67	2	184	2	0.66	N	95	167	84.2	52	154	3.21	Y	N	N	N	N	N	N	0

144	lakshmi	57	F	CSA	Y	Y	N	22.1	77	130	80	77	1	154	1	0.54	N	96	149	68.6	45	177	3.31	Y	N	N	N	N	N	N			N
145	Adhikesavan	50	M	CSA	N	N	Y	23.4	79	110	70	75	N	189	2.5	0.58	Y	102	170	86.8	55	141	3.09	N	N	N	N	Y	N	N	13		
146	Rajan	61	M	AWMI	N	N	N	22.6	75	100	78	70	N	153	1.7	0.76	N	118	167	88.2	43	179	3.88	N	Y	N	N	N	N	N	2.5		
147	Deivasigamani	47	M	UA/NSTEMI	Y	N	Y	23.8	86	108	60	64	1	189	3.6	0.69	Y	179	283	203.4	41	193	6.90	N	N	N	Y	N	N	Y	69		
148	Prince david	49	M	CSA	N	Y	N	24.1	91	128	84	68	1	275	2.2	0.72	N	124	169	87.8	44	186	3.84	N	N	Y	N	N	N	N	8		
149	Saminathan	63	M	AWMI	N	Y	N	23.7	91	150	90	45	1	264	4	1	Y	131	192	103	36	265	5.33	N	N	N	N	Y	N	N	28		
150	Venkatesan	49	M	AWMI	Y	N	N	22.8	89	122	76	40	N	201	3.7	0.88	Y	205	166	89.4	45	158	3.68	N	N	N	Y	N	N	N	16		
151	Prakash	44	M	UA/NSTEMI	Y	N	N	21.5	79	110	86	56	2	244	5.3	1.3	Y	196	291	212.8	51	136	5.70	N	N	N	Y	N	N	Y	33		
152	Elumalai	45	M	AWMI	N	N	Y	26.5	99	138	78	50	1	512	7.4	1.15	Y	68	231	153	34	220	6.79	N	N	N	Y	N	Y	N	82		
153	Lakshmipathy	64	M	CSA	Y	Y	N	23.1	74	100	70	60	1	123	2	0.77	N	96	182	99.2	53	149	3.43	N	Y	N	N	N	N	N	0		
154	Chandra	49	F	UA/NSTEMI	N	Y	N	22	81	104	74	62	N	138	2	0.51	N	106	260	174.8	55	151	4.72	Y	N	N	N	N	N	N	0		
155	Hanif	74	M	UA/NSTEMI	Y	Y	N	23.5	86	156	104	60	2	434	4.6	0.79	Y	164	176	101.4	40	173	4.40	N	N	N	Y	N	N	N	34		
156	Damodharan	51	M	IWMI	N	Y	N	23.9	93	160	88	55	2	287	6.5	1.1	Y	136	265	167.2	37	304	7.16	N	N	N	N	Y	N	N	74		
157	Vijayakumar	38	M	AWMI	N	N	Y	22.1	91	110	78	45	1	338	4.9	0.93	Y	121	268	186.8	48	166	5.58	N	N	Y	N	N	N	N	8		
158	Gurunathan	67	M	CSA	Y	N	N	24.2	87	170	86	78	2	306	6.2	0.92	Y	241	222	131.6	54	182	4.11	N	N	N	Y	N	N	N	58		
159	Jamal	56	M	UA/NSTEMI	Y	N	N	23.6	85	110	84	63	N	123	3.6	0.88	Y	193	257	173.2	55	144	4.67	N	N	Y	N	N	N	N	40		
160	Susanna	57	F	CSA	Y	Y	N	24	92	140	100	66	1	142	4.2	0.81	Y	123	278	199.6	36	212	7.72	N	N	Y	N	N	N	N	40		
161	Kulanthaisamy	68	M	AWMI	Y	Y	N	25	97	132	96	36	2	396	3.9	0.68	N	220	183	102.8	39	206	4.69	N	N	N	Y	N	N	N	14.5		
162	Manikam	55	M	IWMI	N	N	Y	23.5	86	150	90	43	N	154	3.8	0.86	Y	78	255	170.4	59	128	4.32	N	N	N	N	Y	N	Y	33		
163	Anwar	60	M	CSA	Y	N	N	24.6	101	128	78	68	1	264	4.5	0.92	Y	138	288	203.6	34	252	8.47	N	N	Y	N	N	N	N	32		
164	Murugan	50	M	UA/NSTEMI	N	Y	Y	22.2	72	120	74	59	1	129	1.6	0.61	N	110	177	88.6	62	132	2.85	Y	N	N	N	N	N	N	0		
165	Durairaj	60	M	IWMI	Y	N	N	26.4	91	100	70	47	2	289	5.4	1.06	Y	192	271	191	42	190	6.45	N	N	N	Y	N	N	N	26		
166	Abdul Kadhar	48	M	UA/NSTEMI	N	N	Y	20.6	78	130	88	56	N	155	3.8	0.9	Y	90	159	74.6	56	142	2.83	N	N	N	Y	N	N	N	16		
167	Ganapathy	71	M	CSA	Y	Y	N	21.8	83	140	96	68	1	240	3.3	0.81	Y	166	194	109.2	57	139	3.40	N	N	N	N	Y	N	N	20		
168	Selvam	53	M	AWMI	N	N	Y	22.5	78	108	78	40	N	124	2.3	0.64	N	80	183	98.4	47	188	3.89	N	Y	N	N	N	N	N	1		
169	Parthasarathy	28	M	AWMI	N	N	Y	23.4	83	100	80	38	1	285	4.5	0.57	N	95	167	83.4	54	148	3.09	N	N	Y	N	N	N	N	10		
170	Kodeswari	49	F	CSA	N	Y	N	20.2	77	114	84	64	N	118	2.7	0.55	N	105	166	80	48	190	3.45	N	Y	N	N	N	N	N	1		
171	Rajendran	52	M	IWMI	N	Y	Y	20.7	76	138	96	45	N	145	2	0.77	N	112	185	102	46	185	4.021	N	Y	N	N	N	N	N	1.5		
172	Mary	60	F	UA/NSTEMI	Y	Y	N	23.1	83	144	90	58	2	248	5.7	0.6	N	230	257	176.8	44	181	5.84	N	N	N	Y	N	N	N	52		
173	Loganathan	45	M	CSA	N	Y	N	22.8	88	170	100	68	2	291	4	0.78	Y	109	270	184	35	255	7.71	N	Y	N	N	N	N	N	4		
174	krishnamoorhty	46	M	AWMI	N	N	Y	24.5	89	134	80	48	N	167	4.6	1.18	Y	99	248	158.6	37	262	6.70	N	N	Y	N	N	N	N	10		
175	selvaraj	52	M	UA/NSTEMI	N	Y	N	25.2	89	134	88	50	1	440	4.1	0.81	Y	70	189	108.6	41	197	4.60	N	N	N	Y	N	N	N	48		
176	Alimabanu	57	F	CSA	Y	Y	N	22.6	84	140	80	67	2	218	6.1	0.6	N	116	182	97.8	55	146	3.30	N	N	Y	N	N	N	N	5		
177	Subramani	74	M	CSA	Y	N	N	28.5	104	136	84	76	1	168	5.8	0.97	Y	186	190	109.6	43	187	4.41	N	N	Y	N	N	N	N	40		
178	Antony	65	M	UA/NSTEMI	N	Y	N	25.8	95	144	84	63	1	390	7.1	1.21	Y	90	204	118.8	36	246	5.66	N	N	N	N	Y	N	Y	64		
179	Mari	59	M	UA/NSTEMI	N	Y	Y	23.4	85	130	68	65	1	145	1.1	0.63	N	68	252	168.2	56	139	4.5	N	Y	N	N	N	N	N	0		
180	Devarajan	45	M	AWMI	N	N	N	21.7	80	120	70	42	1	321	5	1.07	Y	88	254	171.8	54	141	4.70	N	Y	N	N	N	N	N	3		
181	Ameen sherif	70	M	IWMI	N	Y	N	24.1	99	132	66	48	2	406	3.7	0.86	Y	105	195	117.4	40	188	4.87	N	N	N	N	Y	N	N	30		
182	Sankareswari	63	F	CSA	Y	N	N	23.6	84	128	80	66	N	163	6.2	0.67	N	141	193	103.8	43	231	4.48	N	Y	N	N	N	N	N	2.5		
183	Venkateswaralu	46	M	IWMI	N	Y	Y	22.6	83	130	70	50	1	283	2.2	0.64	N	123	261	182.6	42	182	6.21	N	N	Y	N	N	N	N	10		
184	Jothi	48	F	CSA	N	Y	N	21	80	150	84	68	2	123	5.7	1.22	Y	92	279	200	50	145	5.58	N	N	N	N	Y	N	N	36		
185	Tamina bee	55	F	CSA	Y	Y	N	20.2	79	130	86	73	2	196	3.3	0.68	N	112	281	191.8	48	206	5.85	Y	N	N	N	N	N	N	0		
186	Sridhar	55	M	IWMI	N	N	N	21.9	76	140	82	40	N	174	2.9	0.52	Y	102	248	168	40	200	6.20	N	N	N	Y	N	N	N	48		
187	James	69	M	IWMI	Y	Y	Y	24.2	92	134	86	69	3	392	6.5	1.3	Y	268	268	186	48	170	5.58	N	N	N	Y	N	N	N	64		
188	Wilson	46	M	CSA	Y	N	N	22.4	84	136	78	61	N	133	3	0.49	N	161	203	115.4	54	168	3.75	Y	N	N	N	N	N	N	0		
189	Srinivasan	31	M	PWMI	N	N	Y	29.6	101	120	80	50	1	312	5.1	1.14	Y	130	281	183	30	340	9.36	N	N	N	N	Y	N	N	46		
190	Varadharajan	69	M	CSA	Y	N	N	23.6	94	130	70	68	1	172	6.4	0.95	Y	194	176	97.4	41	188	4.29	N	N	N	N	Y	N	N	44		
191	Padmaraj	50	M	AWMI	N	Y	N	24.4	96	140	90	41	1	261	3.6	0.7	N	106	187	110.2	42	174	4.45	N	N	N	Y	N	N	N	13		

192	Manimuthu	54	M	AWMI	Y	Y	N	26.8	103	154	84	43	2	282	4.4	1.06	Y	256	306	229	43	170	7.11	N	N	N	N	Y	Y	N	99
193	Sampath	65	M	IWMI	Y	N	N	22.5	89	110	70	44	1	233	4.8	0.88	Y	199	265	180.8	56	141	4.73	N	N	Y	N	N	N	N	32
194	Kesavan	65	M	UA/NSTEMI	Y	Y	N	24.6	85	106	74	60	2	404	6.9	1.13	Y	310	178	96.6	52	147	3.42	N	N	N	Y	N	N	N	41
195	Murali	48	M	IWMI	N	N	Y	21.6	84	110	84	50	N	182	3.1	0.58	N	92	191	112.4	46	163	4.15	N	N	Y	N	N	N	N	12.5
196	Kareem	40	M	IWMI	N	Y	Y	31.3	108	140	70	40	2	342	5.1	1.08	Y	87	300	203.4	39	288	7.69	N	N	Y	N	N	N	N	32
197	Shanthi	46	F	AWMI	N	Y	N	21.8	83	138	100	37	1	190	3.6	0.99	Y	76	290	205	36	245	8.05		N	Y	N	N	N	N	20
198	Jeenath kowsal	56	F	CSA	N	Y	N	22.3	76	142	78	66	2	232	6.4	0.68	N	109	170	83.4	58	143	2.93	N	Y	N	N	N	N	N	1
199	Venkatesan	48	M	AWMI	Y	N	N	23.5	89	104	70	42	1	192	3.8	0.48	N	220	175	96	47	160	3.72	N	N	N	Y	N	N	N	5.5
200	Shamugasundram	52	M	AWMI	N	N	Y	23.6	86	130	76	46	N	179	3	0.64	N	89	228	146.2	54	139	4.22	N	N	N	Y	N	N	N	8
201	Amirthavalli	63	F	UA/NSTEMI	Y	N	N	21.7	77	110	84	58	1	112	0.5	0.46	N	210	190	105.6	58	132	3.27	Y	N	N	N	N	N	N	0
202	Ramamoorthy	55	M	IWMI	Y	Y	N	26	96	150	90	41	2	321	6.4	1.16	Y	198	253	160.6	38	272	6.65	N	N	N	Y	N	N	N	40.5
203	Ranganathan	70	M	AWMI	N	Y	N	21.5	81	130	70	37	2	132	4	1.1	Y	78	247	161	44	210	5.61	N	N	N	Y	N	Y	N	68
204	Balaraman	42	M	IWMI	N	N	Y	23.6	88	106	84	48	1	252	6.6	0.84	Y	101	255	173.2	53	144	4.81	N	N	Y	N	N	N	N	8
205	Radha	58	F	CSA	Y	Y	N	22	86	120	84	73	2	278	3.2	0.66	N	143	183	102.8	52	141	3.51	N	N	N	Y	N	N	N	14.5
206	Lalitha	52	F	AWMI	Y	Y	N	23.3	84	148	80	46	2	304	4.8		Y	168	206	120.6	38	237	5.42	N	N	N	N	Y	N	N	48
207	Srinivasan	31	M	IWMI	Y	N	N	23.7	86	130	72	41	1	143	4.9	0.77	Y	276	181	101.8	44	176	4.11	N	N	Y	N	N	N	N	8
208	Rani	58	F	CSA	N	Y	N	21.9	85	130	80	69	1	103	3.8	0.92	Y	90	192	108.4	38	228	5.05	N	N	N	Y	N	N	N	29
209	Dastagiri	42	M	AWMI	N	N	Y	23.9	85	152	78	45	1	153	6.8	0.88	Y	108	148	71	41	180	3.60	N	N	N	N	Y	N	N	30
210	Mohan	50	M	CSA	N	Y	Y	25.1	93	140	90	70	2	342	6.3	0.63	N	96	198	119.8	40	191	4.95	N	N	N	Y	N	N	N	40
211	Balaraman	50	M	CSA	N	Y	N	23.7	87	130	100	65	2	135	3.8	0.74	Y	110	172	91.4	51	148	3.37	N	Y	N	N	N	N	N	4
212	Bavani shankar	58	M	UA/NSTEMI	Y	Y	N	26.6	96	146	96	59	2	365	7.5	1.2	Y	290	283	204.8	39	196	7.25	N	N	N	Y	N	N	N	61
213	Radha krishnan	50	M	UA/NSTEMI	N	Y	Y	23.2	78	124	82	61	1	266	0.8	0.54	N	81	291	207.8	46	186	6.32	Y	N	N	N	N	N	N	0
214	Saravanan	49	M	IWMI	N	N	N	24.5	92	106	74	43	N	137	1.8	0.64	N	102	179	97	54	140	3.31	N	N	Y	N	N	N	N	8
215	Francis Xavier	47	M	AWMI	N	Y	N	21.1	78	130	70	34	1	165	3.6	0.78	Y	120	161	80.6	36	222	4.47	N	Y	N	N	N	N	N	3
216	Srinivasan	50	M	AWMI	N	Y	Y	24	95	144	74	45	1	371	6.8	0.98	Y	108	252	166	38	240	6.63	N	N	Y	N	N	Y	N	80
217	Jayaraj	49	M	UA/NSTEMI	N	N	Y	24.2	96	112	78	60	1	155	5.8	1.21	Y	78	270	189.8	40	201	6.75	N	N	Y	N	N	N	N	40
218	Rajeswari	60	F	CSA	N	Y	N	23.5	80	124	84	70	N	139	1	0.44	N	65	168	85.2	49	169	3.42	N	Y	N	N	N	N	N	0
219	Venogopal	45	M	UA/NSTEMI	N	N	Y	22.4	76	108	78	61	N	113	0	0.63	N	95	186	104.6	51	152	3.64	Y	N	N	N	N	N	N	0
220	Jayapal	62	M	CSA	Y	Y	Y	21.9	74	160	90	65	2	312	5.4	0.81	Y	178	192	109.2	56	134	3.42	N	N	N	N	Y	N	N	66
221	Mohd. Sahil	43	M	AWMI	N	Y	Y	20.8	83	140	90	44	2	154	3.2	0.66	N	90	206	124.8	50	156	4.12	N	Y	N	N	N	N	N	1
222	Tamim Ansari	49	M	AWMI	N	N	Y	23.1	84	130	80	42	N	195	1.1	0.52	N	80	170	89	48	165	3.54	Y	N	N	N	N	N	N	0
223	Manoharan	47	M	IWMI	Y	N	N	24.4	89	110	94	46	1	336	6.7	0.92	Y	234	216	136.8	44	176	4.90	N	N	N	Y	N	N	N	36
224	Sudharshan	65	M	CSA	Y	Y	N	22	85	100	84	76	N	190	2.8	0.61	N	123	284	201.2	52	154	5.46	N	Y	N	N	N	N	N	4
225	Varadharajan	43	M	UA/NSTEMI	Y	N	N	24.6	103	120	78	60	1	219	5.4	0.86	Y	75	208	128.4	45	173	4.62	N	N	N	Y	N	N	N	14
226	Janagaraj	35	M	UA/NSTEMI	N	N	Y	22.9	85	120	70	61	N	193	0.6	0.7	N	88	182	96.2	54	159	3.37	Y	N	N	N	N	N	N	0
227	Desappan	40	M	IWMI	N	Y	N	24.2	90	140	78	44	1	341	3.5	0.68	N	116	161	80.4	44	183	3.65	N	Y	N	N	N	N	N	3
228	Srinivasan	48	M	AWMI	Y	Y	N	22.8	87	130	94	45	1	304	4.5	0.96	Y	228	251	171.4	42	188	5.97	N	N	Y	N	N	N	N	14
229	Vijaya	48	F	IWMI	Y	Y	N	23.9	89	140	86	50	1	291	3.8	0.79	Y	235	177	93.4	48	178	3.68	N	N	N	Y	N	N	N	20
230	Babu	55	M	AWMI	N	Y	Y	20.8	73	130	74	40	1	288	3.3	0.83	Y	95	184	104	42	190	4.38	N	N	N	Y	N	N	N	39
231	Govindharaj	65	M	CSA	N	N	Y	21.5	81	120	80	66	N	154	1.1	0.62	N	78	158	53.8	48	281	3.29	Y	N	N	N	N	N	N	0
232	Karthikeyan	40	M	UA/NSTEMI	N	N	Y	22.4	80	150	88	61	N	165	2.6	0.65	N	106	192	108.2	54	149	3.55	N	Y	N	N	N	N	N	2
233	Sheik masthan	61	M	UA/NSTEMI	Y	Y	N	27.8	98	120	84	60	2	277	7.6	0.98	Y	244	212	121.6	38	262	5.57	N	N	N	N	Y	N	N	74.5
234	Palnivel	37	M	AWMI	N	N	Y	25.2	91	110	70	41	1	246	4.8	0.94	Y	105	296	207.8	38	251	7.78	N	N	N	Y	N	N	N	82
235	Savithri	57	F	UA/NSTEMI	N	N	N	22.7	78	136	84	65	N	168	1.5	0.55	N	96	211	121	42	240	5.02	N	Y	N	N	N	N	N	1
236	Sekar	58	M	UA/NSTEMI	Y	Y	N	21.8	84	130	80	56	2	199	5.5	0.94	Y	161	166	84	54	140	3.07	N	N	N	Y	N	Y	N	89
237	Windsor stanley	64	M	IWMI	Y	N	N	22	88	160	82	47	1	286	7.3	1.04	Y	280	240	161.6	36	212	6.66	N	N	Y	N	N	N	N	38.5
238	Megala	44	F	UA/NSTEMI	Y	Y	N	24	81	140	90	65	2	426	5.5	1.21	Y	118	290	211	45	170	6.44	N	N	N	Y	N	N	N	109
239	Kirubakaran	59	M	CSA	Y	Y	N	27.7	97	140	94	70	1	144	4.7	0.77	Y	124	168	96.4	40	158	4.20	N	N	N	N	Y	N	Y	47

240	Raju	55	M	CSA	N	N	Y	22.1	77	120	80	68	2	297	4.4	0.62	N	86	276	185.4	59	158	4.67	N	N	Y	N	N	N	N	8
241	Periyasamy	62	M	CSA	N	Y	N	20.2	79	150	90	65	1	366	5.5	1.2	Y	110	234	153.6	53	137	4.41	N	N	N	N	Y	Y	N	116
242	Nainabai	57	F	CSA	Y	Y	N	23.2	90	110	80	68	2	109	4.2	0.85	Y	154	176	96.2	51	144	3.45	N	N	N	N	Y	N	N	30
243	Shankar	59	M	UA/NSTEMI	Y	N	N	21.8	78	100	80	65	1	287	3.6	0.93	Y	165	181	98.8	52	151	3.48	N	N	N	N	Y	N	N	20.5
244	Mohd Yusuf	67	M	IWMI	Y	N	Y	22.5	79	144	74	49	N	167	0	0.64	N	245	165	83.4	53	143	3.11	Y	N	N	N	N	N	N	0

Key: M = Male, F = Female, Y = Yes, N = No, DM = diabetes mellitus, HTN= hupertension, WC= waist circumference, EF=ejection fraction,

DD=diastolic dysfuntion, EATT=epicardial adipose tissue tickness, CIMT = caroitid intemis media thickness, COMB = combined test positive of EATT and CIMT,

RBS=random blood sugar, CHL=cholesterol, LDL-C=LDL cholesterol fraction, HDL-C=HDL cholesterol, TGL=triglycerides, TC/HDL=total cholesterol- HDL chlosterol ratio.